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The Medical Clinics of Chicago

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The Medical Clinics of Chicago

VOLUME 1

NUMBER 3

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

TYPHOID FEVER, WITH FULL DISCUSSION REGARDING TREATMENT

I WANT to show you a patient who has typhoid because I want to discuss with you the question of treatment. I thought I would take up with you today somewhat systematically the treatment of typhoid fever.

I am going to pass over some of the things which are so well known and fixed as to make them hardly suitable for discussion. In regard to the prophylactic treatment of typhoid, no two people can disagree. In regard to the care of the body, clothes, and the excreta we can't get up very much disagreement. I want to discuss with you especially the dietetic treatment of typhoid fever. It is an extraordinarily important topic. Twenty years ago I think it is safe to say that the majority of physicians kept their patients on a strictly milk diet. I was taught to believe that anything else than milk was rank poison to the patient. He was given milk and nothing else, and not much of that. The result of this sort of treatment was that the patient simply loathed to see the nurse coming down the hall with a glass of milk. He became sick and tired of it. As a result the appetite became impaired and he took just as little as he could, and when he got through he was in wretched nutrition. It became almost impossible to feed them, and patients developed all sorts of complications. We had great difficulty in keeping their mouths clean, bed-sores were common, and we had many cases of furunculosis, and they often got up emaciated in a marked degree.

I saw a great many more cases of typhoid as an intern, in Cincinnati, than I do now, and I made up my mind that when I got through my apprenticeship I would do something besides feed patients on milk alone. Little by little physicians became more liberal in their diet, but the majority of men up to five or six years ago kept their patients principally upon milk, giving them perhaps other fluids. This was a little better, but during the last four or five years the fashion has swung around the other way. Men are recommending food in quantities—a good deal more than the ordinary diet contains. A man will recommend as much as 4000 or 4500, or even 5000 calories of food a day, which is as much as a man doing the hardest kind of work can consume.

I have always opposed the simple milk diet and the simple fluid diet, but I am just as much against the overfeeding. I see no reason why a patient that is running a fever should be fed more than he would if he were up and about. I have never put a patient on a pure fluid diet, but I don't believe in overfeeding either. In the first place, I think a diet consisting of about 2800 or 3000 calories is just about right. That is just about what most of us consume in a day. Is it necessary to keep the typhoid patients on a liquid diet? Emphatically No. The idea was that by keeping the patient on a strictly milk diet the fecal residue would be less, but that is not true. A man on a strictly milk diet will have hard fecal masses just as often as those on a more liberal diet.

In the first place, I allow them milk if they like it; if they are fond of milk I often give them half cream and half milk. Our ordinary city milk is not so rich that this is unpalatable. Along with this I give them a lot of milk foods, and I am particularly partial to the ice cream preparations. One can prepare many different dishes, such as the parfait, the ordinary mousse, and the fruit ices. I see no reason why ice cream should not be taken in any form unless it contains large quantities of fruit. I think ice cream should be used much more freely than it is; it is palatable and nearly every patient will take it. Then we have the egg foods. They can be given raw, beaten up with milk, soft boiled, poached, or coddled. Fried or hard-boiled eggs are a

little too indigestible for an ordinary patient, but a well-made omelet is very good, and I allow a patient to have one once a day if he likes it.

And then we have the cereals. Any soft cereal can be given. I allow my patients cream of wheat, oatmeal, or anything so long as it is pretty thin. It should be taken with plenty of cream and sugar. I use milk-sugar instead of cane-sugar. It is not so sweet as the cane-sugar and, consequently, a patient will take more, and in this way get a good deal of nourishment. In the same way, in a milk-punch or egg-nog you can put in a good deal of milk-sugar without making it too sweet. I do not know who first suggested this, but it is useful.

Then I want to recommend to you the use of soups, but in recommending soups, I don't mean what are commonly called "slops." Don't give the patients mutton broth or beef-tea. The amount of nutrition contained in these is a negligible factor. The best soups are the cream soups, and they can be prepared in a very delicious way. One can even use as heavy a vegetable soup as the cream of pea. These should all be strained through a coarse kitchen towel. I think the cream soups are much too little used; a cream soup, especially if it be given in a cup with whipped cream on top, will make a very delicious food which most patients will take greedily. I don't object at all to potato if given in the form of a purée. A small amount of well-mashed, creamed potato is allowable. Various things can be given in the form of purées.

Jelly preparations can be given, such as wine jelly, or the ordinary tapioca mixtures flavored with different fruits. My experience is that a good cook and housewife is almost as essential to the treatment of a case of typhoid fever as the physician. The thing to do in treating a patient with typhoid fever is to see that he comes through in some sort of decent nutrition. It is not much of a compliment to a physician to have a patient come through a fever if he has lost 25 or 30 pounds and it takes him a year to get back into good nutrition.

The dietetic treatment is all important. I am sure that I have seen patients literally starved to death—their nutrition so

poor that they fell victims to some of the numerous complications. The diet I have outlined gives you a very liberal *ménu*, and we can still add to it several other things. When asparagus is in season I allow them just the tips of fresh green asparagus or the tips of cauliflower; I see no reason why a patient should not have a spoonful of cauliflower tips if he gets none of the fibrous part.

Will you give them meat? I have never quite seen the necessity for giving the typhoid fever patients meat. If I did give them meat I would prefer to give them grilled sweetbread. This is probably the most delicate meat we have, and a little bit of sweetbread or calf's brain would be the best to give them. I don't like to do it, though, for I feel they are unnecessary. I do not regard a patient as a machine, nor do I think we have to put so much fuel into his boiler every so often. You surely will not forget that the whole trend of investigation tends to show *that the food must be made appetizing*. Simply to put food in his mouth isn't the proper way to do. You must make every bit of food that you give him appetizing; make him glad to get it. If the patient just dreads to see the nurse coming with some nourishment, it generally means that there is something wrong with your management of the case. A typhoid fever patient does not have much appetite, but the food can generally be made appetizing, and you should see that it is.

Now, about the intervals of feeding. They should be short. I generally prefer every three or even every two hours, beginning when he wakes up in the morning and continuing until he goes to sleep at night. I don't feed them at night. The only exception I would make to this would be if the patient was so emaciated that I thought nourishment more important than sleep. But if the patient wakes up during the night I always try to have the nurse get him to take some food, perhaps a little egg-nog, which is quickly made and may help the patient to go off to sleep again.

Feed the patients, then, frequently. Select from these foods a decent variety. Don't just say to the patient or to the nurse you can have any of these things that you like. It is frequently a good plan not to let the patient know what he is going to get.

Give him a variety each time, and you will find that on this sort of diet your patient will come through in good nourishment and there will be much less chance of complications than we used to have.

I ran across a statement of Shattuck, and it corroborated my own experience to such an extent that I repeat it. It is to the effect that we don't see so much falling out of the hair as we used to following this disease. I confess that it did not occur to me that this might be a matter of nutrition, but Shattuck attributes it to the fact that our patients come through the disease now in a much better state of nutrition.

Now I want to call your attention to another thing: A great many men seem to think that when the temperature is coming down to normal that means that the intestinal ulcers have healed. Not at all. A great many men who think this keep their patients on a low diet until the temperature comes down and then feed them anything. I believe that is a mistake. I don't make any change in my diet until the temperature has been normal for two weeks, and if you can keep a patient in bed for this long a time and keep him on this diet all the way through, you will frequently find there has been very little loss of weight. Not long ago I took care of two very severe cases of typhoid, both with hemorrhages, on this diet, and because of the severity of the symptoms I kept them in bed for three weeks after the temperature disappeared, and they got up with a loss of weight of only about 5 pounds apiece and feeling in excellent condition.

Do not make a hard-and-fast scheme for feeding every patient, whether it be a high calorie or any other kind of diet. The patient must be kept in nutrition. I do not recommend giving large quantities of food at a time, but give it to them frequently. I do not recommend the use of 4000 or 5000 calories of food, for that is altogether too much; it is more than he would consume normally, and I can't sympathize with the present fad of overfeeding.

Now I want to take up the question of the antipyretic treatment in typhoid fever. Ever since the Brand bath has been advised everyone has been busy trying to escape it. The Brand

bath, as you know, is very difficult to give and requires the assistance of a couple of attendants. I have never been able to warm up to the Brand bath as a good many men have done, and I think it is safe to say that it is now losing in favor. I don't believe that old statement in the text-books that when the temperature exceeds 102° or 103° F. the patient should be put in a bath. I don't believe that the temperature in itself needs to be combated. I think the good that comes of a Brand bath is not on account of the reduction in temperature. I think they are given much less frequently than formerly, and I hope if I ever have typhoid and one of you are called upon to treat me you will not give me the Brand bath as a matter of routine. When a patient is just a little stuporous he had best be given nothing more than just a sheet rub. He should be wrapped in a sheet, sprinkled with cold water, and rubbed. If the stupor becomes more pronounced he can be given a tub-bath right in bed. That is what we do here. Put a rubber sheet under him, pour water over him, and rub. Don't forget that the important thing in any hydrotherapeutic measure is the rubbing. The action of the cold, of course, is to contract the peripheral capillaries, and you rub them so as to keep the capillaries well dilated. One man has stated that he has seen patients killed by being put in a cold bath without any rubbing. I have never seen this, but the patients should certainly be rubbed vigorously. A mild case should be treated by the sheet rub and a very severe case by making a tub of a rubber sheet. A still more severe case should be treated with an ice rub. The patient is covered with a sheet and rubbed with a block of ice. In severe stupor a good thing is to pour ice-water on the head from a little height. Take a pitcher of ice-water and pour it on the head from a distance of 14 or 16 inches. Patients who are somnolent, breathing badly, and can't be brought out of their low, muttering delirium in any other manner, can be brought around very quickly in this way.

The temperature in itself does not need any treatment. We don't begin to worry about a temperature of 104° F. as we did a few years ago. I don't regard the temperature in itself as an indication for interference unless very high.

Now a word about the use of antipyretics: My advice is like that given by Puck to the man who was going into matrimony—"Don't." However misplaced that advice may be on the subject of matrimony, it is certainly sound in regard to antipyretics. They should not be used in any case of typhoid.

One very important point that I might have discussed under the subject of diet is the question of fluid. I have rather strong ideas on this subject, and agree with the men who think they should have all the fluid they can hold. I try to lay it down as a rule that a typhoid fever patient should take water until he passes double the amount of urine that he did before he started treatment. If he was passing 1 liter of fluid a day, give him fluid until he passes 2 liters or more. The point in allowing him this large amount of water is this: it certainly dilutes the urine and the toxins, and I think that we have a smaller number of complications when we give large quantities of fluid than when we don't. One or two authorities have expressed the fear that there is some danger of dilatation of the heart in this. That I think is a purely theoretic conception. Give him just plain, common water, or give him any of the simple spring waters that he may happen to prefer. Give him fluid in the form of lemonades, or fruit juices, but give him water, and water, and more water until he consumes at least 3 or 4 liters a day.

Another question is, Will you give your patients tea and coffee? I see no reason why they should not have tea if they feel that they will be uncomfortable without it. I like to give it to them with considerable quantities of milk-sugar, for it aids nourishment. If they are very susceptible to coffee I give them the preparations weak in caffein, such as Merck's Dekofa.

Now the question of alcohol in typhoid: Will you give alcohol? As a routine, No. If your patient is an alcoholic and there is chance of his developing delirium tremens, then give him a sufficient quantity of alcohol to avert it. If I had a patient in whom heart failure was developing I would give him alcohol. That doesn't often happen, and the consequence is that I don't often use alcohol in typhoid fever, but if a patient likes it and is accustomed to taking it I have no objection to giving 1 ounce or

1½ ounces of whisky in egg-nog daily, but to recommend it as a routine I think is a mistake.

As to drug treatment: I have a little "specific" that I give to every case, a little diluted hydrochloric acid. I give this because it can't possibly do any harm. Most patients have a little deficiency of hydrochloric acid, and if it does no good, it is a pleasant placebo; it gives the patients the idea that something is being done for them, and possibly it takes the place of the hydrochloric acid in the stomach. With one exception, this is the only drug I ever use as a routine. I usually give a little hexamethylenamin; I think by giving this drug in small doses we can prevent the development of urinary complications. I think it is valuable in every case toward the end of the disease to make sure that the bacilli no longer exist in the urine. Given in fair doses, say 7 or 8 grains two or three times a day, I think it is very useful. Outside of these two things I don't think of any drug that is needed in the average case.

Now there are one or two complications that ought to be considered. The first is bed-sores. Don't get them; they are a reflection on the nursing. Change the position of the patient frequently. Don't let him lie in one position any longer than if he moved himself. The body should be rubbed well every day or twice a day. The buttocks should be thoroughly cleansed every time the patient soils himself, and be thoroughly rubbed as well.

The most dreaded complication is a perforation, and to my mind the first thing to do is to call a surgeon and have the abdomen opened. It is better to open an abdomen once in a while and find no perforation than to wait twenty-four hours and find that you did have a perforation, but that a general peritonitis has set in.

Now a word or two about the symptoms of this complication: I recommend that in every case you have a leukocyte count made twice a day and take the blood-pressure twice a day if perforation seems to threaten. Why? The leukocyte curve in typhoid is down; at the end of the third week it may be only 3000. The blood-pressure curve is down; instead of having 120 you will

probably have 100. The first symptom of perforation is a rise in the blood-pressure, so you should take this at least every day so as to be able to recognize a sudden rise in the blood-pressure. This, accompanied by pain and tenderness over some part of the abdomen, are the first signs of perforation. The leukocyte count goes up, and this is a point that is ordinarily overlooked. It may go up to 6000 or even to 9000 and not be any more than it was when he was normal, so keep a close watch of the leukocyte count and the blood-pressure so as to be able to recognize any rise. A patient who has only 2500 or 3000 leukocytes, when the count runs up to 6000 has a real leukocytosis.

Will you give them morphin. I don't. Put on hot compresses if you like; that will make the pain bearable and will not mask the symptoms.

Another serious complication is hemorrhage, and one of the things I do is to watch the stool with the Weber test. If you find that the patient is having a little blood in the stool, and every day he is getting a little bit more, that will warn you that he is on the verge of a larger hemorrhage, and will make you a good deal more careful. What will you do when you have a hemorrhage? That is a disputed point. In my judgment the first thing to do is to give them a good big dose of morphin. I would ease up on the feeding for twelve or twenty-four hours. If the hemorrhage is sufficient to amount to anything and the pulse is becoming weak and thready, the thing to do is to give the patient adrenalin. It does not raise the blood-pressure and some men have objected to its use on the ground that it might force out the clot. A suitable dose is 1.0 to 1.5 c.c. of a 1 : 1000 solution hypodermically. Perhaps the best way of all to give it is in normal salt solution, $\frac{1}{2}$ to 1 liter, subcutaneously. If the hemorrhage is very great, the salt solution may be repeated, and the foot of the bed can be raised. The adrenalin may be repeated in five or six hours if necessary.

I have only had an opportunity of giving adrenalin in a few cases, and it is difficult to tell just what its effect was. Many cases where I have given only morphin have gone through just as well.

Remember that there is no such thing as an abortive treatment of typhoid. Keep the patient on the diet that I have recommended for at least three weeks after the temperature is normal; give him 2800 to 3000 calories of food. Do not give him solid food as soon as the temperature drops. Under this treatment a patient should get up in fairly good nutrition. Give him all the fluid that he can take, preferably enough to increase the urine up to 3 liters a day. Hydrotherapeutics should be utilized, sheet rubs for mild cases, cold tubs in more severe cases, ice rubs in still more severe cases, with ice-water poured on the head. Drugs should not be given as a routine, except perhaps hydrochloric acid in small doses. Hexamethylenamin is useful in all severe cases. Lastly, remember that it is always a reflection on your treatment if the patient gets all sorts of complications or loses much weight.

LEAD-POISONING. FINDING THE LEAD LINE WITH A HAND LENS. INNUMERABLE SOURCES OF IN- TOXICATION

THE first patient today is a Lithuanian, thirty-eight years of age, who comes in with the following history: He is constipated, has pain in the abdomen, complains of vomiting, loss of appetite, headache, sweats, weakness, and itching of the skin. He never had anything like this before. He is a day laborer. The onset occurred about two weeks ago, when all the symptoms gradually developed; he thinks the itching developed first, beginning on the inside of the thighs, gradually ascending and involving the sternum, then the thorax and then the arms; it has been quite intense, distressing enough at night to interfere with his sleep. This lasted about a week and then gave way to real pain. The pain is largely in the belly, radiates to the back, goes to the chest, sometimes in the cardiac region, and sometimes radiates down the thighs; it is very intense; it has been knife-like in character and has spread around from the center of the abdomen. He then became con-

stipated and the constipation has been of fairly high grade; he thinks if he did not use a cathartic the bowels would not move more than once in three or four days. Ten years ago he had an attack of constipation which lasted three or four days.

The vomiting occurred with the pain, has not been constant, usually occurs in the morning. The sweats he has been having off and on; they have not been very severe, but every now and then he has one so severe that he has to change his clothes.

His personal history is nearly negative. He drinks some beer and a little whisky. The family history is negative and venereal history negative except for one or two attacks of urethritis some years ago. These symptoms are all of a more or less indefinite character, and it would be pretty hard to pick out of this group anything that would claim our attention immediately.

His physical findings on admission can be summed up in a very few words: He is well developed, muscular, has lost no great amount of weight; the head, eyes, ears, and throat are negative, tongue clean and moist; the chest shows nothing abnormal except a slightly ringing aortic second sound, from which we inferred that his blood-pressure was up a little bit, and we found the blood-pressure to be 148—slightly increased.

The abdomen at the present time is rather soft and relaxed, there is some tenderness on deep pressure all over the abdomen, but not in any particular place. I think you will obtain the same impression I did, that the pain is not really severe. He is more afraid of being hurt than he is actually hurt. There is no free fluid in the abdominal cavity; in short, nothing abnormal about the abdomen. The liver and spleen are normal, his reflexes are normal. The temperature has not exceeded 99.2° F. since he came in. The blood examination shows 3,800,000 reds, 4800 whites, 55 per cent. hemoglobin—a moderate anemia. If we assume 5,000,000 reds to be the normal count, he has only a moderate degree of secondary anemia.

Now, the question is, What suggests itself? The man comes in with these vague general symptoms, lasting a couple of weeks, with no temperature, the symptoms I enumerated above, physical findings absolutely negative except for a slight increase in

the blood-pressure, nothing to amount to anything. Tenderness all over the abdomen, not very marked, however, and a moderate degree of anemia. The urine is negative, acid in reaction, no albumin, no cells, no casts. What things suggest themselves to you?

VISITOR: An afebrile typhoid.

DR. WILLIAMSON: Hardly; what else would you think of?

VISITOR: Tuberculous peritonitis.

DR. WILLIAMSON: Tubercular peritonitis is, I think, out of the question, but he might have some form of intestinal parasites. What else?

SECOND VISITOR: Chronic lead-poisoning.

DR. WILLIAMSON: What makes you think of chronic lead-poisoning, or was that just a guess?

SECOND VISITOR: Has any differential blood-count been made?

DR. WILLIAMSON: The differential shows nothing particularly abnormal. Who will make another suggestion?

THIRD VISITOR: Was the Widal negative?

DR. WILLIAMSON: No Widal has been made yet. Let's take it up from this standpoint: all that the man complains of are general symptoms; they point to no particular place, except perhaps the gastro-intestinal tract, but they occur so commonly that we did not think of them as evidences of gastro-intestinal involvement, for most of the acute diseases come down with them.

In the first place, it has been suggested that it might be afebrile typhoid. He has been in only thirty-six hours and so far as we know he has had no fever whatever. He has no enlargement of the spleen, no rose spots, no looseness of the bowels, but is constipated, and in the absence of all these things there is nothing to point to typhoid.

Let us pass on to the subject of intestinal parasites: they produce anorexia; they will produce vomiting at times, may even produce pain in the abdomen, might possibly produce sweats and weakness. The only answer to that is to find them, and we have not found any. Examination of the stool has been nega-

tive, so we can say as far as we have gone we have seen nothing to indicate the presence of parasites.

Some one suggested plumbism. With what symptoms is lead-poisoning generally associated? With very much the same as we see here. What diagnostic sign do we find in lead intoxication? Burton's line. The characteristic line on the gums. This is pathognomonic of plumbism, so we will look carefully and see if we can find anything that might be interpreted as a lead line on the gums. A superficial examination would lead us to believe that there was nothing, but when we look closely we will see between the incisor and the canine a little bluish spot, and if we examine this with a hand lens we will see that we have a very typical and very characteristic lead line.

When examining for the lead line you should place the patient so that the light falls across the teeth and then look very carefully with a hand lens, and what do you see when you find a lead line? There are two lines that are commonly described as the lead line: one in the angle between the gum and the teeth, right down on the teeth in the angle. This can be found in any man who works in any dirty, metallic dust, and can be removed by thorough cleansing of the teeth. All this means is that the man works in some dusty, metallic occupation. This is a false lead line.

The true lead line occurs *in the gum*, and in its incipency is very often overlooked. Now what do you see? Not a line, strictly speaking, but *little bluish spots*, hard to recognize with the naked eye, but with a lens we can see fifteen or twenty of these little spots, and the most common position is in the little papilla of gum that is between the teeth. That is the most frequent place for them. They are so characteristic that when they have once been seen there is absolutely nothing else of which I am aware that simulates a lead line. Not simply a line that looks dirty or bluish, but little, tiny punctate spots in the gum itself. These occur as the result of the decomposition of the lead in the mouth, by the H_2S from the little decomposed particles of food.

Now as to the significance of the lead line: The true interpretation is that *the patient is leaded*, that he has absorbed lead

into his system, and that lead has been taken up and deposited in the tissues of the gum. Does that mean that the patient has lead intoxication? Perhaps not. He may have this lead line and yet not have enough stored up in the system to produce intoxication, but, practically speaking, you will be perfectly safe in thinking that the patient with a well-developed lead line has a lead intoxication. I want you all to become thoroughly familiar with the lead line because it has such great diagnostic value. There is no getting away from it. It is just as significant as the determination of the malarial parasite.

A few words about these symptoms. Are they symptoms of lead intoxication or are they incidental? They are the typical symptoms that occur in a lead intoxication—these vague general symptoms. The only one of those symptoms that does not seem to me to be definitely associated with the lead intoxication is the itching. Itching is not usually associated with lead intoxication.

Symptoms.—Then let's take up the symptoms one by one and see how they occur. The ordinary case usually appears with a simple anemia. Every now and then the presence of a simple anemia suggests to us the possibility of lead intoxication. Next comes the constipation, which becomes very marked: the bowels do not move for several days, the patients do not respond to salts very well; they require larger doses than usual. Then the so-called lead-colic appears. This is usually preceded by symptoms of a more or less indefinite character. Patients will generally tell you that they have had little twinges of pain before the onset of the real colic. The colic itself is a pain that is sharp and lancinating in character, starting a little below the navel and radiating all over the abdomen. If the pain is sufficiently severe it may even be referred outside of the abdomen down into the thighs. How long does the lead-colic last? Sometimes only two or three hours, sometimes two or three days, and now and then two or three weeks. Two weeks is not an unusual time to have a patient suffer with a severe lead-colic. While the pain is at its height the abdomen is tense, hard, and contracted. The pain, as a rule, is relieved by pressure, and so we find that patients will fold their arms over their abdomen and press hard for the



Fig. 83.—The lead line as seen on the gums with the unaided eye and under low magnification. (Drawn from life.) (Case of Dr. Charles Spencer Williamson.)

purpose of relieving the pain. The pulse is nearly always characteristic, slow, and of high tension. The combination, therefore, of a hard, contracted belly, a slow pulse, and somewhat increased tension, with no fever, should always be suggestive of lead intoxication. The triad of symptoms embraced by the anemia, the constipation, and the colic makes an almost certain diagnosis of lead intoxication, and when we have added to this triad the lead line *in the gums* we can make the diagnosis with perfect certainty.

I want to say something about the sources of lead. It is a tremendously important subject. As little as 1 or 2 milligrams of lead in the system may give rise to symptoms in some individuals. Where do these persons get it? The painter is a classic example; the plumber is another classic, but the plumber does not get it as often as the painter because he does not work with lead in a powdered form. The most serious lead intoxications occur in the white-lead factories; this industry is probably responsible for the most serious lead intoxications that we have.

In the last few years another group of industries has produced a large number of intoxications. I refer to that large body of men engaged in sandpapering various articles made out of wood; such as carriage beds, automobiles, etc. The lead paint is rubbed off, it gets into the air in the form of dust, and they absorb the lead. This is one of the largest sources of intoxication.

Another source of infection is through the rubbing down of some of the fillers. In the various arts many fillers are used which are sandpapered down, and the worker absorbs the dust with which the air becomes charged.

I think it would be interesting to you to read the very latest thing which has been published on this subject, an article by Dr. Emery R. Hayhurst, who is now with the survey of the State of Ohio. The State has appointed a commission to investigate occupational diseases, and one of them is lead intoxication. The first sentence of this article is very instructive. It says that if the sandpapering and rubbing down of paste fillers were stopped the large number of cases of lead intoxication would be greatly decreased, even to one-tenth the present number. The vast army of men who work where automobiles, carriages, etc., are made

furnish a great many cases. This volume has only reached me during the past week, and it represents an actual tabulation of the workers in one community.

It is the man *who does not apparently work in lead* who is most frequently affected; it is the man who sandpapers painted wooden articles who gets it most often. It is particularly important to know that the men who paint indoors or who sandpaper indoors are more likely to acquire it than those who work outdoors where they do not absorb so much dust. We see it in the lead miners and we see it in men who do not know that they work in lead. I saw lead-poisoning last year in two cases in men who drove an ordinary junk wagon. They drove a team and would go out where they removed the brass from faucets, etc., and when I investigated I found that there was always a certain amount of lead attached to the brass, and this large amount of lead rattling around in the wagon produced lead dust, and they both had very serious cases of lead intoxication. I have seen a number of cases in girls who worked in soldering establishments. This solder contains a very high proportion of lead, and every now and then we find a worker in this substance who has a severe plumbism.

The sources of lead are almost innumerable. One in particular I can't help recounting because it occurred in a man whom I know quite well. He was a gunsmith, and when I asked him if he worked in lead he asked me scornfully if I thought guns were made of lead. But finally I made a visit to his shop to investigate what he did, and I didn't spend more than half an hour before I detected the source of his lead. In putting a fine gun in the vise, in order to avoid defacing the gun, they face the vise with lead, and this soft lead holds the gun in its grip without scratching its surface. After fastening the gun in the vise he would work over it, and every now and then as he got down close to the surface of the vise his file would cut off a little bit of the lead, which, of course, made lead dust, and he got this on his hands.

There are three methods of getting it into the body. No matter where it comes from, it is by one of these ways: either the respiratory system, the gastro-intestinal system, or through

the skin. The last of the three is a negligible factor. The majority of it is taken in through the respiratory system or the gastro-intestinal system.

I can't help saying a word in regard to prophylaxis. To my mind, from what I know personally and what I have seen in reviewing the literature, the most important thing is thorough cleansing of the hands. If the painter would scrub his hands so that he got rid of every bit of lead and then removed his clothes with the paint upon them he probably would never get lead intoxication. This experiment has been made. Have the worker scrub his hands clean and then immerse them in a pail of water containing sodium sulphid, and even when you think they are perfectly clean you will get a black color, showing that some of the lead is still there. This is a very important test. When you are doubtful about the hands being entirely clean, have the patient scrub up and then use such a solution to be sure that it is all removed.

How frequent is lead intoxication? The number of men who work in lead is increasing every day. How many of them are ever intoxicated? A census was made recently of 100 house painters and 59 showed evidences of lead intoxication, and yet painters are not nearly so liable to it as those who work indoors.

Thanks to the automobile industry, we are making a large number of storage-batteries, and the consequence is that we have a great many workers in these factories who show lead-poisoning. There are a number of pigments and various preparations in the paints of which the formula is not given and the worker does not know they contain lead. These are only a few of the salient points. There are so many ways in which lead can be taken into the system that they are well-nigh innumerable, but these are the most important. The men who rub down lead paints, the painters themselves, and then the men working in storage-battery factories, are the every day cases, the white-lead factories giving the largest percentage of serious cases.

There are two or three other points I want to speak of in connection with the diagnosis. One is the change in the blood. In the beginning when the case is progressive we find baso-

philic degeneration. This consists in the development in the erythrocytes of granules which stain with various basic stains. This develops in all sorts of conditions. It is found very commonly in pernicious anemia and occurs in the beginning of acute lead intoxication. It does not occur with anything like the same frequency when the case has become chronic. I recommend to you the study of these basophilic granulations. The nicest stain to bring it out is the thionin stain. The granulations are stained an intense blue. One other point is of importance, the so-called Liebermann test. This has for its purpose the determination of the hemolyzing power of the red blood-corpuscles. If you drop blood into water it undergoes hemolysis; in hypotonic sodium chlorid solution it does the same. Strangely enough, in these cases of lead intoxication the resisting power of the corpuscles is increased, and we find that they will not disintegrate in a solution of sodium chlorid until below the usual strength, which is 0.45 down to 0.40 per cent.

I think I neglected to state that the lead line is by no means always present. It occurs in probably half the cases of plumbism. When it is present it makes the diagnosis absolutely certain, but when it is absent it does not prove that lead intoxication is not present.

To sum up: The patients come to us first complaining of anemia, headache, constipation, anorexia, vomiting, and sometimes dizziness; a good deal of weakness, lassitude, and then twinges of pain in the abdomen, and later on actual definite attacks of colic. These are the symptoms occurring in the majority of cases. In about 1 case in 2 or 3 we have the lead line developed in the early stages. The resistance test is valuable as confirmatory evidence.

Now let me urge upon you the great necessity of diagnosis in these early cases, the great importance of looking over the teeth very carefully with a lens, going over them tooth by tooth, and you will be surprised to see how often just a few little dots will settle the diagnosis for you. They will often get away from you when just a few of the vague, general symptoms are presented; so let me urge upon you to look for these cases when you have a cer-

tain set of symptoms. Take the trouble to inquire from the patient or some fellow-worker whether he comes in contact in any way with lead in his work, since there are so many opportunities for lead to get into the human system. I may say that when we put down this man's occupation as a painter it was not what he said he did; he said he was a laborer, but we found that he was engaged in painting most of the time.

ANOTHER CASE OF PLUMBISM

Now I have another case that I want to show you. This man is a German, a laborer, thirty-nine years of age. He came into the hospital saying that he was taken sick three weeks ago. The first thing was lack of appetite. He was markedly constipated. Along with this were cramps in the upper part of the abdomen. Since that time the cramps have been almost continuous and for the past three weeks he has scarcely been free from them; he is very weak. This has shown itself in the arms as well as in the legs. Two weeks ago he had one or two attacks of vomiting, but none since. After the initial attack of cramps the pain settled down into a dull ache in the stomach and since then he has had almost continuous dull pain rather than cramps. For the past two days he has noticed pain on urination, and says the stream has been rather difficult to start. His past history is fairly characteristic. He has been working in a lead smelter for the past month. Previous to that he worked in concrete. He has only worked in lead for a month, and then out in a lead smelter where they have been producing pig lead from the ores. His history in every other respect is negative. Only a little over two weeks from the time he entered the lead occupation he was seized with this cramping pain and vomiting, the cramps later giving way to dull, continuous pain. He has had no rise in temperature at any time; it has not been above 98.6° F. since he entered the hospital.

The physical examination disclosed nothing about his head, the chest showed nothing abnormal, the blood-pressure was 150; blood examination showed 4,100,000 reds, 7600 whites, the differential count shows nothing abnormal; the basophilic degeneration is pretty well marked. The urinalysis was negative, and that's all. In the abdomen in this case we have findings a little different from the other patient. Here the abdomen is hard and rigid, but in the hardness and rigidity we do not see a peritoneal involvement,—not the kind of soreness we see in an appendiceal abscess. At once you are struck with the way the abdominal walls are contracted. There is no enlargement of the liver or spleen. The pulse in this case has not been slow with any degree of consistency; it has been 68, 66, 70, but occasionally goes up to 100, and I take it that this is when he experiences the most pain.

Now we want to look at his gums carefully. In spite of the fact that he has been employed in lead only a month he has a much more definite line than the other man. The dots are so close together that they form a real line. They appear over the upper teeth in front, but on the lower they are not so pronounced. I want you to notice particularly the gum next to the left lower incisor and you will see a beautiful little string of dots. Occasionally these patients develop a stomatitis which eats away the edge of the gum, and every now and then patients come in with this so marked that the lead line is destroyed. The symptoms in this case bear a very striking resemblance to those in the previous case. You might think that because the lead line is so pronounced in these two cases that we always find it, but, as I said before, we do not. In the cases observed by such a commission as the one referred to on occupational diseases, they find the lead line occurs in only about one-half the patients. You gentlemen who are out in general practice see these cases earlier than we do in the hospital service, and you must not think that the lead line is always present in the beginning.

This last case is very instructive to my mind because the patient never worked in lead until a month ago, and yet he has a lead line there which looks as if he might have worked in lead for

years. There is another striking point in connection with lead intoxication. Some patients will develop it within a very short time after they are exposed and others will work in it for twenty or thirty years; then they sometimes come down suddenly with all the symptoms of severe intoxication. They develop, as you know, a very interesting group of palsies, the so-called wrist-drop and the so-called foot-drop, sometimes a typical Duchenne's type of paralysis. But this is by no means so common as the colic. I should say that the colic was about ten times more frequent than the paralysis. Of the more serious symptoms of lead, then, we find the colic the most common, occurring much more frequently than paralysis. Every now and then you find a patient who will come down within a few days. I once knew a man who worked only seventy-two hours in a white-lead factory and developed a typical lead encephalopathy. In the long run all the workers in lead become affected more or less; that is, the continuous worker in lead nearly always acquires lead intoxication sooner or later. This is one of the great problems that we have to face in our American commonwealth, and it will take us some time to get on a par with our European neighbors. Abroad in all the parish hospitals in one great city there were but 13 cases of lead intoxication in a year. I don't know how many we average here, but I, personally, see more than 13 cases a year. When we stop to think of the fact that out of 100 house painters 59 showed intoxication you will see how common it is.

Now one last point: When the workers have no colic and no paralysis they think they have no need to worry about it, but the worst effects are more far reaching. Lead has a very definite effect upon the arteries. First it has an effect upon the intima of the arteries and then upon the middle coat. We see the so-called lead mesarteritis where we see the arteries much involved. This lead mesarteritis we are sometimes capable of recognizing clinically. The arteries feel a little different from an arteriosclerosis. When you see a young man with arteries that resemble a bicycle tire which is only partially inflated, if you obliterate the pulse above the wrist you can feel this very nicely. With diffuse resilient thickening of the arteries it is nearly always due to lead.

So always examine the arteries and see what the condition is. This patient shows it very nicely. If you obliterate the pulse above the radial and then palpate the artery you will get it very easily.

That leads us to the effects upon the heart. The thickening of the media and the blood-pressure, which remains high, lead ultimately to hypertrophy of the heart. Then, too, we have the typical lead kidney. I had one of the most typical examples I have ever seen in a patient who worked in a pottery and who died as the result of kidney involvement. The specimen from his kidney is a very typical lead nephritis due to chronic intoxication. So you see the remote effects of lead are very striking, and one is its connection with gout. Here in America lead is tolerably active in connection with gout. Quite a respectable number of cases of gout have as one of the etiologic factors a chronic lead intoxication. We have very serious and very wide-reaching consequences from lead intoxication. So when a man comes to you who says he does not care anything about the lead line on his gums, because he has no colic and no paralysis, when you come across such a patient, insist upon his undergoing treatment, because it is quite impossible to say how much his kidneys or his heart may be damaged.

Now just one last word: A chronic lead intoxication may go along for many years without any symptoms, but when they do develop they develop very quickly and with great intensity. A patient with a plumbism may go along with practically no symptoms until he develops a broken-down heart or a lead nephritis, and they are very hard to handle. It is very difficult to eliminate the lead from the systems, but you should do everything in your power to get rid of it as early as possible.

TREATMENT OF LEAD-POISONING

Prophylaxis.—My idea of the treatment of lead-poisoning is identical with that which I once heard given by a distinguished surgeon in regard to bed-sores, namely, "Don't get them."

In the overwhelming majority of instances lead-poisoning can be prevented, and as good citizens it is the urgent duty of all

of us to use our best efforts to have laws passed and *enforced* looking toward the prevention of industrial plumbism. This is hardly the place for me to take up with you the scope of such laws, but I will show you a copy of the placard issued by the Ohio Commissioners, and which is supplied to manufacturers upon request. These instructions contain in a nutshell the most important points to be observed by the employee to prevent lead-poisoning. I want to emphasize particularly the second recommendation. The avoidance of dust is, in my judgment, the most important single point, and the second is to prevent the access of lead to the system by means of dirty fingers.

NOTICE

INSTRUCTIONS TO EMPLOYEES HOW TO PREVENT LEAD-POISONING

(1) All workers exposed to lead dusts, lead fumes, lead solutions, and lead compounds are liable to poisoning. These poisons get into the body through the nose while breathing, or through the mouth when chewing, or swallowing, or wetting the lips.

(2) Do all you can to keep down dust. When sweeping or cleaning always dampen with water, oil, or wet sawdust. Where dust cannot be kept down, you must wear a respirator. This must be cleaned out at least once a day.

(3) Eat breakfast before going to work. Drink milk at meals and, if possible, once between meals. Do not eat meals in workroom. Leave workroom at meal times.

(4) Keep dirty fingers out of your mouth, and off of your food and whatever goes into your mouth. Wash hands, arms, and face with warm water and soap before eating, going to the toilet, or quitting the workroom. Clean your lips and rinse out your mouth before eating or drinking.

(5) A mustache, if worn, must be kept short. Do not wear a beard. Keep finger-nails clean and cut short, also loose skin about the nails or hands.

(6) Do not chew tobacco or gum while at work. Avoid the use of intoxicants in any form, as they promote lead-poisoning.

(7) Take a full bath with warm water and soap at least twice a week.

(8) You must wear overalls and jumpers while at work. Wear a cap if exposed to dust or fumes. Do not wear your working clothes outside of the working place.

(9) Keep your bowels moving, if possible, once a day. Report to your foreman if you notice (1) loss of appetite, (2) poor sleep, (3) indigestion, (4) continual constipation, (5) vomiting, (6) pains in stomach, (7) dizziness, (8) continual headache, or (9) weakness in arms, limbs, or body.

Treatment.—When a patient comes under your observation with a well-developed case of lead-poisoning, what will you do with him? For the colic a full dose of morphin with atropin should be given at once hypodermically, and simultaneously every effort should be made to empty the bowels completely. This is not a small task, since there is obstinate constipation, and it often requires full doses of cathartics to bring about the required results. The best laxatives are sodium or magnesium sulphate, since these, in addition to their cathartic power, precipitate the lead as an insoluble sulphate. Enemas containing magnesium sulphate should also be used freely. One or two evacuations are not sufficient. Free catharsis should be persisted in until the entire bowel has been thoroughly emptied. If the patient's hands are still contaminated, which can be ascertained by the sodium sulphid test above referred to, they should be scrubbed thoroughly, and the finger-nails cleaned so that no more lead may enter the system from this source. Iodid of potassium should be given cautiously, about 5 grains three times a day, since there is some danger of liberating the lead in the system too rapidly.

Lead paralysis is treated along the same general lines as any other peripheral neuritis. Particular care should be taken to avoid the use of alcohol, since it has been found that its use predisposes strongly to lead neuritis.

A TYPICAL CASE OF GOUT, SIMULATING AN INFECTED FINGER

Now I am going to show you another joint case. This man is a teamster, sixty years old, married, and comes in for swelling,



Fig. 84.—Typical acute gout. (Drawn from life.) (Case of Dr. Charles Spencer Williamson.)

pain, heat and redness in the joints, with loss of appetite. Says he has had attacks like the present for the last six years, although this last attack is the most severe. His first attack came on in the great toe and he thought it was a swollen "bunion." He does not remember about an acute onset, but sometimes at night he has been roused from sleep by a pain in the great toe. This would then pass to the other side and eventually disappear. In the course of time the pain passed to the phalangeal joints, and that is the pain for which he came in.

You see before you one of the most interesting hands I have looked at for a long time. The interphalangeal joints of the ring and little fingers are about as badly swollen as anything you can imagine. (See Fig. 84.) I brought him in to show him to you because during a previous attack one of our Chicago surgeons outside the hospital thought he had an acute infection in the joints and opened one of them. Although they are still very much swollen and you can see that they contain fluid, they were much more swollen and tender a week ago than they are now. We want to look at that swollen "bunion" of his. We see something that does look like a bunion on the right phalangeal joint. On the left side you can see again the small joints of the tarsus and metatarsus are greatly swollen and very tender.

Now I don't suppose after showing you several cases of gout that there is any doubt in your minds that this is a very typical, clean-cut case of gout. If you have any doubt, all you have to do is to come up to his ears and see a very choice collection of tophi. The prettiest one of all we have under the microscope. We took it out and put it up in permanent form because it shows the sodium bi-urate crystals very beautifully.

A nice way to preserve all sorts of small crystals is by means of glycerin jelly. All you have to do is to snip off a little piece of the jelly with your knife, put it on a slide, and pass through the flame to melt it, put in whatever you wish to preserve, and place it under the cover-glass. For keeping the various small eggs of intestinal parasites it is absolutely unsurpassed, and the crystals you see under the microscope are put up in this way. You will notice that this specimen shows a bit of cartilage with all these

crystals sticking out of it. There is a laughable side to this case; it is laughable to think that a good surgeon would open up a joint in a gout case, thinking that he had an abscess, and yet as you looked at the joints it would have been hard to convince yourself that they were not going to suppurate. They never do suppurate, no matter how intense they look; they always subside and, of course, nothing surgical should be done.

We have had a really extraordinary run of cases of gout and I could not refrain from bringing this case down. In spite of these enormously swollen joints his temperature has never but once run 100° F. This is quite according to rule for a gout case.

CLINIC OF DR. RALPH C. HAMILL

COOK COUNTY HOSPITAL

NEURITIS

NEURITIS is a subject of particular interest for clinical discussion because any pain is apt to be called "neuritis," with the consequence that inasmuch as pain is the cause for many individuals seeking medical advice, and inasmuch as that pain is so frequently spoken of as neuritis, the differential diagnosis of the various forms of peripheral pain, or "neuritis," is a very complex but important one.

The name neuritis implies an involvement of nerve structure, and has come to be applied particularly to involvement of peripheral nerves. The inflammatory agent may be of many different characters. In the past alcohol was given the place of honor, with overfatigue, occupational strains, cold and exposure, and trauma as contributing factors. Other ingested poisons, or other poisons taken into the body, which were considered as causative agents of neuritis, were very numerous; lead, arsenic, and mercury are among the more important. Of late there has been a tendency to consider these poisons, alcohol included, as being in the category of the contributing factors, such as cold, etc. It has been thought that they act merely as predisposing factors, as factors preparing the way for microbic invasion. It is now known, through the work of the Memorial Institute, among others, that alcohol diminishes the antitoxic faculties of the body—the ability to build defenses against microbic invasion. As examples of this pathogenesis the first case is fairly typical. The two cases before us this morning are typical of a large number seen in this hospital.

Case I.—B. K., aged thirty, a pressman by occupation, entered the hospital in September, 1914. He had been suffering

for eighteen days with pain in the calf muscles. For two weeks these pains persisted, though he continued his work, which called for excessive use of the muscles of the lower leg. Finally he was obliged to give up his work and found great difficulty in even getting about on his feet. About the time he began to have the pains in the legs trouble with digestion occurred, with vomiting. Shortly before entrance to the hospital he noticed that there was blurring of objects and letters, coming on soon after beginning to read.

Two years ago he had a similar attack, with pains in the legs and some weakness, although this attack was not sufficiently bad to confine him to bed. This previous attack came on shortly after a Neisserian infection.

The family history is negative. The alcoholic history is striking; for several years he has been an excessive drinker of beer, taking also six to seven drinks of whisky a day.

At the time of entrance he was unable to stand owing to weakness and pain in the legs; there was a congestion in the pharynx; the teeth showed a marked pyorrhea; but there was no particular cervical adenopathy. He complained bitterly of severe pain in the calf muscles, into the nature of which we will go later.

On physical examination the most striking feature was the bilateral foot-drop. He was entirely unable to walk. There was complete paralysis of all the muscles of the leg. The feet were very cyanotic, cold, and clammy.

Seven days after entrance the patient developed a marked tremor of the hands, insomnia, restlessness—all signs of an incipient delirium tremens, which, however, cleared up in the course of the next five days. After about three weeks the weakness of the hands practically disappeared.

Upon entrance the temperature showed rather marked variations, ranging from 99.2° to 97° F. in the course of one day; on another it was 98.2° F. in the morning, 99.6° F. in the afternoon; again 98° F. A. M., 99.4° F. P. M.

Since the time of entrance there has been slow, gradual improvement in the general condition. He still has a sort of a dull, aching discomfort, especially after any effort. Upon examining

him, as you see, the foot-drop is very pronounced (Figs. 85 and 86). The great toe is flexed toward the sole. The foot is at an angle of about 150 degrees with the line of the leg. The feet are distinctly cyanotic; they are rather cold and clammy. There is a marked flattening in the peroneal region, almost a groove between the tibia and fibula. The calf is also quite atrophic. When he

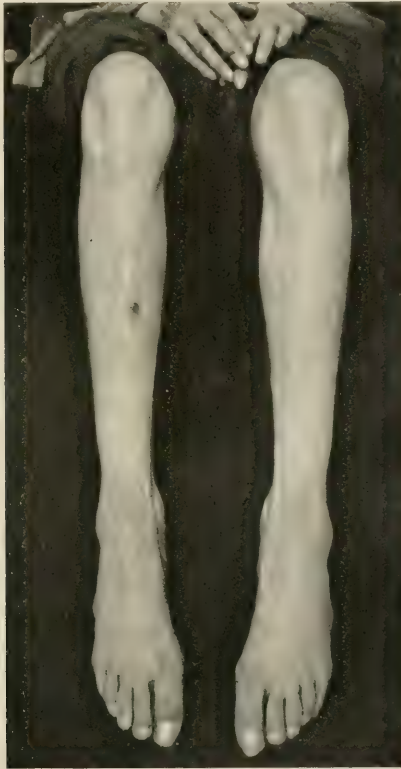


Fig. 85.—Foot-drop in multiple neuritis.

attempts to flex the foot dorsally the only movement is a slight inversion and adduction due to action of the *tibialis anticus*. Efforts at plantar flexion of the foot are fairly strong, although, because of the shortening of the *tendo achillis* with the consequent extended position of the foot, the movement is through a small arc only. Extension of the leg on the thigh is considerably weaker

than normal; flexion is fairly strong. Passive movements of the feet and legs are all possible, though passive dorsal flexion is somewhat restricted through shortening of the Achilles tendon.

The grip is fairly powerful, considerably stronger in the right than in the left hand. Flexion at the wrist and elbow and movements of the shoulder-joint are of normal power. Extension

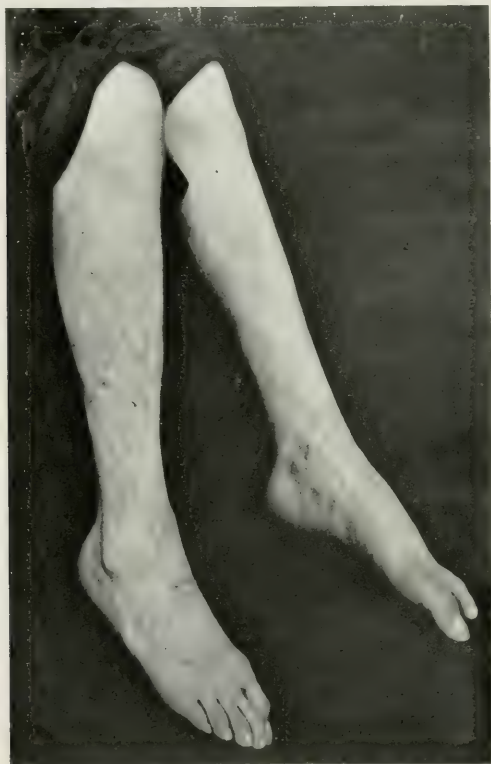


Fig. 86.—Foot-drop in multiple neuritis.

of the wrist, especially on the left side, is considerably enfeebled. Passive movements are normal in the arm.

The deep reflexes in the legs are lost: knee-jerk and Achilles' jerk. The wrist-jerks are decreased, especially on the right side. The jaw-jerk is normal; the cremasteric reflexes are diminished; the abdominal also. Blood-pressure and heart are normal;

mentality seems normal. There is at present a well-marked cervical adenopathy, a marked gingivitis, pus can be expressed from the gums of the lower jaw in several places.

When we come to the sensory examination, we find a condition quite typical of peripheral neuritis; a condition the presence of which is perhaps the most pathognomonic sign of involvement of the peripheral nerves. It is perhaps not quite true to speak of it as pathognomonic of involvement of the peripheral nerves, since the same condition is found in disease of the posterior root ganglion. Light touch is not well appreciated on the lower legs. Pricks with the pin are at first not appreciated, but after four or five jabs he complains bitterly of a deep, burning, continuous pain, diffuse rather than definitely localized. On the other hand, what is perhaps the most striking feature of the sensory disturbance is the hyperesthesia, the hypersensitiveness to deep or even moderately deep pressure. This is strikingly brought out when in trying to obtain a plantar reflex the fingertip is drawn along the foot sole (using moderately deep pressure only) there is, after a moment's pause, a bitter complaint of pain. You notice that single pricks with the pin are not appreciated.

This combination of a diminished or lost response to sharp superficial stimulus, with an increased, exaggerated response to deep, ordinarily painless stimulus, is a characteristic finding of peripheral neuritis. It is a factor of the greatest importance in the differential diagnosis.

Case II.—The second case to be demonstrated is that of this colored man, thirty-five years of age. He entered the hospital July 21st, complaining of shooting pain and numbness of the extremities, incontinence of urine, and some difficulty in walking in the dark. These troubles had begun about four weeks previous to his entrance, at which time he suffered from shooting pains down the legs, and the numbness developed to a degree that made his feet feel like pads.

In his previous history the only disease of importance is a gonorrhea a year ago. He is married, his wife is living and well and has had two miscarriages; no living children. He has been drinking to excess for about five years.

Two years ago he had an attack of trouble which he calls "rheumatism." He couldn't walk because of the pain, which was "same as I have now"; he was unable to work for about four months, and during that time there was more or less difficulty in controlling his water. These are the only facts of importance in his history.



Fig. 87.—Foot-drop in multiple neuritis. Fig. 88.—Foot-drop in multiple neuritis.

In the examination you see this very lean colored man who does not appear to be very sick. You notice, however, that when he sits up with his legs hanging over the side of the bed his toes hang down in a line at an angle of about 150 degrees with the line of the lower leg (Figs. 87 and 88). There seems to be a very marked relaxation of the structures around the ankle-joint

permitting of such extreme extension, an extension practically impossible for the normal.

As he extends his arms you notice that the hands hang down at a right angle with the forearms. Such a drop or such an angle can only be accomplished through extreme voluntary action of the flexors in the normal. If you look at his legs more carefully you see that the wasting is quite marked below the knees. The thighs are fairly well preserved. The same discrepancy is apparent between the forearms and arms. The wasting in the legs is especially apparent in the peroneal group. The normal rounding which extends outward from the line of the shin bone has entirely disappeared; instead of a convex line we have a concave line; there is a trough instead of a ridge between the anterior borders of the tibia and fibula. The forearms, though wasted, do not show nearly the amount of change apparent in the legs. There is some straightening of both the radial and ulnar borders, however, and the space between the dorsal borders of the radius and ulna is much less full than normally. Passive movements in the arms and legs are all easy, with the exception of forcible dorsal flexion of the feet. It is impossible to flex them beyond a right angle, and even that causes an exclamation of pain due to stretching of the calf muscles.

Voluntary movements are good in the arms, the shoulder girdle, the pelvic girdle, and the muscles of the thigh. There is marked weakness in extension of the left hand, somewhat less of the right. His grip is very weak on both sides. Dorsal flexion of the toes is practically gone on the left side, entirely on the right. You notice, if any manipulations are made of the forearms or the legs, which require stretching of the muscles or any considerable pressure upon them, all such manipulations are more or less painful.

In the sensory examination we see the same disturbance as shown in Case I: Loss to touch and pain over the inner lower shin and ankle; also a small patch of analgesia and anesthesia on the dorsum of the left foot and another small patch on the anterior part of the right ankle. The lines of the touch disturbance are more extensive than those of the pain. In marked contradis-

tion to this loss of superficial sensibility is the marked hyperalgesia and complaints of pain when even a gentle stroking along the foot sole is made with the end of the finger. This is especially true on the left side. This corresponds to the complaints already spoken of—of the tenderness elicited upon manipulations.

Upon testing the deep reflexes we find that whereas the Achilles reflexes are entirely absent on both sides, the knee-jerks are brisk. The wrist-jerks show a marked difference: the right is rather brisk, the left is weak and appears entirely confined to the body of the biceps muscles rather than to the supinator longus, as is usual. The biceps and the triceps jerks are brisk, the right stronger than the left. The pupils are normal and the cranial nerves are normal.

Since his entrance into the hospital he has run a slight fever off and on up to the present time. At first there were many times when his temperature was recorded at 100° or 100° F. plus a few tenths. Of late he occasionally has 100° F.; more frequently he varies between 98° and 99.6° F.

Upon entrance the urine showed albumin and a few casts. A lumbar puncture done shortly after entrance showed six cells to the cubic millimeter, the fluid exuding under increased pressure, but the tests for globulin, the Nonne and Ross-Jones, were both negative. Alveolar abscesses and pyorrhea with considerable cervical adenopathy, especially on the right side, are of extreme importance and will be spoken of when we come to the causes of the condition.

Before discussing at length the etiology and diagnosis of these cases I should like to present a third, because of its similarity and yet fundamental differences. It was so similar that for three months the patient has been recorded and considered as multiple neuritis by the house staff.

Case III.—This man is fifty years of age, a watchman by occupation, who ten weeks before entrance into the hospital, on May 21, 1915, began to complain of heaviness of the feet. There were pains in his legs, and the hands began to pain and were so weak as to be useless. He gradually became worse, his hands were somewhat swollen and burned. The feet and legs were so

weak as to be unable to support him. A point which should have attracted the attention of the intern making the examination and diagnosis was that there was no particular tenderness of either the arms or legs. The picture of the disease was dominated by the weakness rather than by pain.

In his past history it is of great interest to learn that he had his right foot operated upon for club-foot when three months of age. You will notice that the right leg is shorter and smaller than the left, and that the right foot is also shorter and smaller than the left. This difference is probably due to an early poliomyelitis or polio-encephalitis, and by "early" I do not exclude intra-uterine. He is married, his wife is living and well; he has six children. He had a Neisserian infection twenty-four years ago. His mother died at eighty-four, of old age, his father at sixty-seven, of diabetes. One brother died of tuberculosis, one of "whisky consumption"; one sister died of cancer of the stomach and one brother was in an insane asylum twice.

So far as his alcoholic history is concerned, he was diagnosed "alcoholic neuritis." He had been drinking three or four whiskies a day up to three months before entering the hospital.

Examination made by the intern at the time of his admission to the hospital showed the pupils equal and regular, though a little sluggish to light and accommodation. The chest examination was normal, the blood-pressure 115 systolic and 90 diastolic. The abdomen was negative. There was a flaccid paralysis of the legs, a pes cavus of the right foot, both arms showed some edema at the wrists and of the hands. There was a little wasting of the forearm muscles and considerable wasting of the arm muscles and the shoulder girdle. The muscle and joint sense, the stereognostic sense, mentality, vision, hearing, and speech were normal; there was no loss of superficial or deep sensibility.

This combination of normal sensibility, no particular tenderness, wasting more extreme about the shoulders than the forearms, should have indicated that the process was other than a multiple neuritis on an alcoholic basis. I said that the paralysis was flaccid, which included or implied a loss of deep reflexes. This was true of all the deep reflexes except the jaw-jerk, which

was normal. The abdominal reflex was also gone. The cremasteric was markedly diminished, as was also the plantar. There was no Babinski.

An examination of the blood and spinal fluid for syphilis was entirely negative. At no time did he run any febrile temperature, except that on a very few occasions 99° F. was recorded. I should have said that the spinal fluid showed an increased amount of globulin in response to the Nonne test. There were no foci of infection discoverable any place; the teeth were unusually firm for a County Hospital case; the sinuses, examined by Dr. Boot, were normal.



Fig. 89.—Dropped wrist in progressive muscular atrophy.

Now as you look at him you notice the same dropping of the foot as in the other two cases shown, much the same dropping of the hands (Figs. 89 and 90). The paralysis is truly flaccid, but the paralysis is of much greater extent than that shown by the two previous cases. It involves the entire extremities. There is the paralysis in the feet and the toes, the hands and the fingers, that you saw in the two previous cases; in fact, it is even more marked. There is also, however, a paralysis of movement of the elbow-joint and the shoulder-joints; also almost complete loss of movement of the knee-joints, and marked weakness in the move-

ment of the thighs. Flexion and extension of the thighs, however, is possible. Adduction and abduction are practically abolished. Passive movements elicit no complaint and are very easily made.

A careful sensory examination reveals no disturbance. As we watch him we occasionally see a slight flicker and fine twitching in the various muscles of the body, perhaps most frequently seen in the small muscles of the hands, as you see at present in the first interosseous of the right hand; now you see the same sort of twitching on the left triceps, and now in the left *teres major* or *minor*.

The electric examination shows interesting findings which aid greatly in the diagnosis. There is a loss of faradic response in all the muscles of the extremities. It produces, however, a tetany of the *rectus abdominalis* and of the *pectoralis minor*, of the *serratus magnus*, and of the *levator anguli scapulæ*. Where the faradic fails to produce a response, the galvanic shows a slow, lazy, or sluggish contraction which in many cases is greater to the make of the positive pole than to

the make of the negative pole. These findings, the loss of faradic response, and the slow galvanic response, more active to the positive than to the negative poles, constitute the typical reaction of degeneration. I should have excepted the left *quadriceps femoris* and the *gluteal* muscles, which show a fair response to the faradic current and a good response to the galvanic.

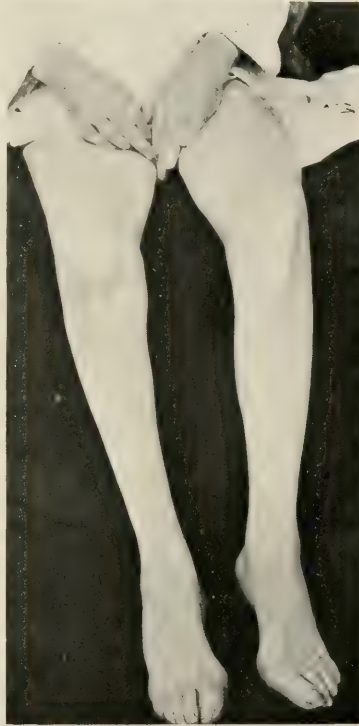


Fig. 90.—Foot-drop in progressive muscular atrophy.

The muscles of the face, the sternocleidomastoid, and the upper portion of the trapezius all show normal reactions. The platysma, on the other hand, shows the reaction of degeneration.

When asked to protrude his tongue you notice a little slowness in protrusion. There is a rather fine tremor running through the muscle, and the upper surface is rather markedly wrinkled. The soft palate hangs quite far forward and you will notice that when we have him take a deep breath or say "Ah," even though he ejaculates the "Ah" as sharply as possible, you will notice that there is a rather slow elevation of the soft palate when you compare it to what takes place in the normal. Other cranial nerves, including the optic nerve, show no variations from the normal.

Cases I and II are cases of true neuritis—a text-book picture of alcoholic neuritis, with the extensor muscles involved more than the flexors, the legs more than the arms, with incipient delirium tremens after a few days in the hospital in one case, and a history of prolonged abuse of alcohol in both. The differential diagnosis is not at all difficult because the picture is quite typical.

Case III might be confused with these two former patients because of the general appearance; that is, there is a foot-drop and a wrist-drop, a paralysis at the extremities of all four limbs. Here, however, the similarity ceases. The paralysis is really more marked in the proximal segments of the limbs, the atrophies are more marked in the same locality, but, more important still, there is a complete lack of tenderness of the atrophic muscles, there is no sensory disturbance and no particular vasomotor disturbance. In the first two cases there are evident foci of infection—in the gums. In Case III there is no such focus of infection. Cases I and II show a slight febrile temperature; Case III does not.

Case III is really an example of progressive muscular atrophy with lost reflexes, fibrillary twitchings, muscular atrophies more marked in the proximal segments of the limbs, and typical reaction of degeneration to the electric tests. I merely presented it because of the fact that as a picture the patient presents the same general features upon superficial inspection as do Cases I and II.

The condition most frequently confused with alcoholic neuro-

tis is locomotor ataxia. In locomotor we have the lost deep reflexes, such as are found in these cases, in locomotor we also have a complaint of severe pains in the legs.

It is respecting these pains that I wish to speak most in detail, because, as I said at the beginning of the clinic, it is these pains, commonly called "neuritis," or it is pain of one sort or another which most frequently leads individuals to seek medical attention. The pains in this case were practically continuous, of a dull, heavy, boring character, with occasionally sharp twinges, practically uninfluenced by any medication other than an opiate, made a little worse by exertion, and persisting over a long period of time with practically no period of intermission. These pains were associated with objective sensory findings which were highly characteristic; namely, the analgesia or hypalgesia to sharp stimulus, with hyperalgesia to pressure. Also—a point which I neglected to emphasize—an extreme tenderness of the calf of the leg to pressure. This combination is absolutely distinctive of neuritic conditions.

The condition found in locomotor ataxia, or the pain complained of, is very different. Ordinarily it is intermittent. The patient complains of a sharp jab, like that of a sharp knife or a needle, then there is complete relief, followed by repeated jabs. These jabs may come once an hour or once every five seconds. Usually after the jabs have been repeated for a few times the area in which they occur becomes somewhat tender to touch. These jabs may run down the length of the leg or, on the other hand, they may be confined to an area the size of a dollar at some point along the leg, most commonly about the knee-joint or about the ankle. Associated with these pains the objective sensory examination shows a diminished sense of pain to both superficial and deep stimuli. Pinching of the calf does not cause pain and, more characteristic, the tendo achillis can be sharply squeezed without causing pain. This difference in the characteristic pains of the two conditions is sufficient in itself to settle the question in the majority of cases.

The other common form of pain in the lower extremities which could be confused with neuritis is due to arthritis. In these

cases it is localized in a joint and surrounding structures, it is present almost exclusively upon movement, the joint structures rather than the muscles that move the joint are tender, and ordinarily the reflexes about the joint are increased rather than diminished, as in neuritis. Atrophies can occur about an arthritic joint suggestive of those seen in these cases which might be misleading, but such atrophic muscles are rarely tender.

In discussing the treatment of such cases the etiologic factors are, of course, of prime, utmost importance. In the first place, there must be complete withdrawal of alcohol. That is true whether one sticks to the old idea that alcohol is the real causative agent, or whether one assumes that the poison is truly microbic in origin, and that alcohol merely breaks down the resistance of the body against the microbic action.

In the first case there is a history of two attacks of neuritis. Before the first attack he had had a gonorrheal urethritis, a condition which cleared up in a fairly short time, but nevertheless constituted a focus of infection. At the time of his second attack, at least at the time of his entrance into the hospital, he had a very bad condition about the gums and teeth. Pus could be expressed from several places in his gums. That this pus, this infection, was threatening the general organism was shown by the cervical adenopathy. This adenopathy is distinctly more marked on the right side—the side where the infected gum condition is the worst. I believe that the latter theory, that of microbic origin, is the correct one in this case of alcoholic neuritis.

A most striking example of the nature of the origin of the disease was presented by a case which died in the hospital about six months ago on my service. It was a case of a young girl, twenty-three years of age, who had had an operation a few months previous to her entrance. She had been in the habit of taking two or three glasses of beer every evening, certainly not more than that amount. At the time of her admission to the hospital there was a typical bilateral foot-drop with characteristic sensory disturbances, lost reflexes, vasomotor disturbance—namely, cold, cyanotic, clammy feet and hands.

Shortly after her entrance into the hospital she developed a

typical delirium tremens, the attack lasting three or four days and then clearing up. During her first few weeks in the hospital she ran a typical, low-grade, infection temperature—a variation of from $1\frac{1}{2}$ to $2\frac{1}{2}$ degrees between morning and evening temperature, and there was discovered a point on the gum between the two lower incisors which looked infected. The mucous membrane of the lower lip opposite this point gave the appearance of having been touched by a stick of caustic. A culture made from this point upon the lip gave practically a pure culture of staphylococcus. A similar culture was obtained from the gum at the point of infection. *x*-Ray examination showed an alveolar abscess at this point—*i. e.*, at the roots of the lower middle incisors. In spite of tooth extraction, which, however, was somewhat delayed, she gradually grew worse, developed decubital ulcers on the heels and over the sacrum, the neuritic manifestations spread to the arms, she lost control of the sphincters, and finally died after about three months.

At the autopsy there was discovered a small abscess in one of the suprarenal capsules. Otherwise there were no particular gross findings. The microscopic details have not yet been worked up.

In this case it is not reasonable to suppose that the two or three glasses of beer could have been the cause of such a disease picture and course. From the autopsy and from the cultures made during life, from the septic temperature, and from the general pictures of the disease it was evident that the trouble was of microbic origin.

In this first case alcohol is the only predisposing factor which we need consider. In the case I told you of, however, there is, besides the small amount of beer taken, also the operation with a certain diminution of resistance of the body consequent to the operation. This factor may be taken to account for the ready surrender to the noxious activities of otherwise innocuous microorganisms.

In both these cases, then, besides withdrawal of the alcohol, the other most important therapeutic procedure is the clearing up of any source of microbic toxins. These microbic toxins are

frequently found in the oral cavity. Their presence there is normal; their activities become abnormal when the resistance of the body against them is lowered. The products of their metabolism then become poisonous. In the case I cited it was apparent that the germs themselves were able to make entrance into the body also, as shown by the suprarenal abscess. In the first case it is impossible to say whether it is merely their toxins or whether the germs themselves also enter beyond the mouth and its dependent lymphatics.

Normally, then, these micro-organisms are present in the mouth. There are various places in the mouth where they can develop undisturbed—the crypts of the tonsils and the edges of the gum are most suitable for their development. Also, when they have once reached a point of relative virulence they are not only present in the mouth, but they are also being constantly swallowed and are setting up various gastro-intestinal disturbances. It is impossible to say how much of their toxins are being absorbed from the intestinal walls at any time. The elimination of any possible source of microbic intoxication, then, is of the utmost importance.

Medically, there is very little to be done other than such palliative measures as opiates for the pains—a dangerous procedure with these more or less unstable people. Heat relieves the pains the most satisfactorily of any of the means which we have at our disposal other than opiates.

Heat can be applied in a variety of ways. Perhaps the most satisfactory combines heat, moisture, and light. This is accomplished by putting the part to be treated between blankets wrung out of hot water, covering them over with a tin tunnel-like arrangement on the under surface of which are two to six electric bulbs, covering the whole over with a couple of blankets; turning on the light, one has a very good hot pack at his disposal. This arrangement applied for twenty minutes will frequently give relief where other means fail. Naturally, however, such an arrangement as this will be found only in hospitals, though it could be put together in a home with the aid of a tinsmith.

Heat can also be applied merely by wringing out sheets and

blankets in hot water, wrapping the part in them, and laying hot-water bags or hot bricks on either side, covering the whole over with dry blankets.

The first patient shows where another therapeutic indication has been neglected. Some one should have flexed his foot dorsally several times each day in order to prevent the contraction of the Achilles tendon which has taken place.

Massage in these cases is sometimes impossible because of the pain it produces. Where the pain does not prevent, gentle massage of the affected muscle groups is important. Electricity can be used after the acute symptoms have subsided. Gastro-intestinal antiseptics, such as salol and beta-naphthol, are to be recommended. After having proper dentistry done where it is necessary, cleansing tooth-powders or mouth-washes are indicated.

For many years strychnin has been recommended in the treatment of neuritis after the subsidence of the acute period. It can be given in doses up to $\frac{1}{30}$ grain three times a day by mouth with perfect safety in an adult. In fact, with careful watching, it is possible to give even $\frac{1}{10}$ grain three times a day. Signs of having reached the physiologic limit are: rigidity of the neck and back, a feeling of drawing in these regions, and an extreme nervousness or irritability. When these signs are observed the dosage can be cut down about one-third and continued for a considerable period of time. With careful watching there is no danger in giving strychnin in this manner.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

LARGE FIBROID OF THE UTERUS PRODUCING CARDIAC AND RENAL DISTURBANCE

WE will show the first patient this morning rather hastily because of her condition. She is thirty-nine years of age, and entered the hospital about three weeks ago with the following history: Shortness of breath and enlargement of the abdomen, swelling of the feet and ankles, and inability to walk. Her illness appeared about six weeks ago with dyspnea; later there was swelling of the abdomen, ankles and legs, and subsequently an inability to walk. The trouble has been constantly progressive.

Past History.—She had a pneumonia in 1895; otherwise previous history is negative.

Family History.—The father is dead, cause unknown; the mother died from an operation; brothers and sisters all living and well. There is no history of tuberculosis or carcinoma.

The temperature has been normal, with the exception of a slight elevation to 99° F. The pulse has varied from 100 to 120; the respirations from 24 to 48. The urinalysis, as recorded here, reveals no albumin and no casts. The blood examination showed a white count of 8200; reds, 4,520,000; hemoglobin, 90 per cent. A differential was normal.

On examination, you will observe that the patient is in more or less apparent discomfort; the respiratory rate we have already noted; her breathing is rather rapid and shallow, and accompanied by an expiratory groan. We also take note of the swelling, involving particularly the eyelids, both upper and lower, with an edema of the conjunctivæ, more marked on the left than on the right side, edema of the face, especially the temporal regions, and

a marked edema of the lower extremities, the feet and legs and the dependent portion of the thighs being very edematous.

The history calls our attention to the condition of the abdomen, which has increased in size since the onset of the illness a few weeks ago. It is irregularly enlarged, more bulging on the right than on the left. On palpation we at once come upon a mass which fills the lower half of the abdomen, extending from one iliac crest to the other. This mass is firm and solid, but irregular; we can outline definite projecting smaller tumor masses. Even on inspection we can locate a distinct bulging at several points; they are rather firmly fixed to the larger underlying tumor. The larger mass is only slightly movable; it is rather firmly fixed.

On percussion, there is flatness over this tumor mass, no fluctuation, with impaired resonance in the dependant portion of the abdomen upon either side. The liver is not palpable nor is the spleen.

On inspection, the apex-beat is in the fifth intercostal space displaced outward to the nipple line. On palpation, the apex-beat is not strong nor forcible; no thrills are present at the apex or the base. On percussion, the cardiac dulness extends two fingers to the left of the nipple line and up to the third rib. On auscultation at the apex, a fairly loud systolic murmur is heard; no murmurs can be heard over the base of the heart; the aortic second is not loud nor accentuated. The blood-pressure, taken at the time of the first examination, was 110 systolic. The further examination of the patient we will not pursue because of her condition.

You have, perhaps, noticed her deep cyanosis and the rather unfavorable general condition, and I will tell you that the examination of her lungs reveals impaired resonance with numerous loud, moist râles over the lower lobes posteriorly. Our concern is particularly centered on the cardiac condition and the findings in the abdomen.

DR. TICE: What is this tumor mass?

VISITOR: A sarcoma or carcinoma.

DR. TICE: Yes; there are several possibilities. First of all, we have to remember the history. The onset, according to the

history, dates back only one month and a half, when she complained of shortness of breath and swelling of the feet, which finally resulted in inability to go about; at that time she noticed that the abdomen was enlarging. The family history is negative. The pelvic examination is also negative for any cervical involvement; there is no history of a hemorrhage or of a vaginal discharge of any kind. We can rather definitely exclude the existence of a carcinoma or a sarcoma. A tuberculous peritonitis? The patient has no temperature, there is little or no fluid in the abdomen.

The outline of this tumor, the irregular, hard, nodular formation, more or less fixed, and, on bimanual examination, definitely associated with the uterus, would strongly suggest a fibroma of the uterus.

The cardiac findings are those of a mitral insufficiency. There is no history, etiologic or otherwise, on which to base an endocardial involvement. The patient denies ever having had a rheumatism or a specific infection. There is only the history of a pneumonia in 1895. There is, however, an edema of the lower extremities, of the face, of the entire surface of the body, particularly of the dependent portions. The urinalysis recorded here is negative, but this was a voluntarily voided specimen and one which was alkaline in reaction, and we must conclude that the specimen had undergone decomposition and was of no value whatsoever. No subsequent urinalysis is recorded, but we feel justified, in view of the cardiac involvement, in making a diagnosis of a chronic interstitial nephritis with a secondary heart. A cardiorenal involvement with multiple fibroids of the uterus.

What influence has this fibroid had in producing her present condition? It is easy to conceive, from the size of this tumor, that it may unfavorably influence the circulation, particularly by pressure on the splanchnic area. Another interesting possibility is the influence of this large tumor upon the renal function by pressure upon the ureters, interfering with the action of the kidneys.

The prognosis is unfavorable; the patient is growing decidedly worse. She is daily becoming more and more comatose.

PERITONITIS WITH ASCITES

DR. TICE: What is the diagnosis?

VISITOR: It is an ascites probably due to a cirrhosis of the liver.

DR. TICE: On what do you base the diagnosis of cirrhosis?

VISITOR: She gives a history of using alcohol moderately and I think the liver is small. The liver is not palpable below the costal margin.

DR. TICE: Could you palpate the spleen?

VISITOR: No, I could not; the abdomen is too full of fluid.

DR. TICE: Is there any collateral circulation?

VISITOR: I could not see any.

DR. TICE: Did you feel a mass in the lower part of the abdomen?

VISITOR: There doesn't seem to be any.

DR. TICE (to a Second Visitor): You feel it; what could that be?

SECOND VISITOR: It might be the bladder in the median line or it might be a tumor.

DR. TICE: What form of tumor? Did you see the other case?

SECOND VISITOR: No, I did not.

DR. TICE: She had a fibroid filling the lower half of the abdomen. Could this be a fibroid?

SECOND VISITOR: Yes, it might be a fibroid or an ovarian cyst.

DR. TICE: This patient is thirty years old and entered the hospital on the third of this month. The history consists of a gradual swelling of her abdomen, which appeared three months ago, accompanied by pain. This was followed by repeated attacks of vomiting, extending over the entire time until the last few days. There is also a history of pain in her back, which appeared simultaneously with the pain in her abdomen. There is also the history of pain at the time of evacuation of the bowels. There is an increased frequency of urination associated with some discomfort. There is no history of dyspnea or cough, and if there has been any loss of weight it has been slight. Appetite is good.

Past History.—She had the ordinary diseases of childhood; an attack of diarrhea one year ago.

Menstrual History.—She was regular up to three months ago; since that time there has been no menstrual period. The family history is negative. Is she pregnant?

VISITOR: We could not elicit such a history.

DR. TICE: The patient was pregnant once and miscarried. She denies a venereal infection. There is a history of very moderate use of alcohol, but this only for a very short period of time and extending back only during the past year.

When this patient entered the hospital her temperature was 101° F., pulse 120, respirations 28; the following day, the 21st, in the morning the temperature was 101° F., in the afternoon 103° F.; on the 22d the temperature in the morning at 8 o'clock was 101° F., in the afternoon 102.2° F.; on the 23d, in the morning, 100° F., in the afternoon 100.8° F.; this morning the temperature at 8 o'clock was 99.4° F., pulse 80, respirations 22.

When the patient was first examined her abdomen was enormously distended with free, movable fluid; no tumor mass could be felt, and a paracentesis abdominalis was done on the 21st, the day following her admission to the hospital; at that time $10\frac{1}{2}$ liters of a straw-colored fluid were removed. This fluid had a specific gravity of 1020; a cellular differential count revealed approximately 86 per cent. polymorphonuclears; a fair number of reds were present. Smears were negative for the presence of bacteria, but cultures were made from the fluid, three all told, and two of them were positive. One, a bouillon culture, was negative; the two blood-agar were positive and growths appeared within ten to twelve hours, and the organisms practically all of the same type, consisting of definite cocci that were Gram-positive.

Now, as to the present condition: The examination of the chest, heart, and lungs I will state reveals nothing. They are absolutely normal. The abdomen is only slightly distended; the patient lies in the recumbent position with the knees flexed. We find the abdomen distended and bulging, particularly in the flanks on either side, with a definite distention in the lower portion, and upon palpation a definite tumor mass is present, which extends up almost to the umbilicus. This tumor mass has

a definite median position and is globular in shape. It is rather soft, only slightly tender, and readily movable. There is absolutely no rigidity of the abdominal muscles, no tumor mass in any other portion of the abdomen; the liver and spleen are not palpable.

On percussion, there is a dulness in the flanks as well as over the hypogastrium. (Patient turns on side.) On change of posture it is now tympanitic where flatness was obtained in the dorsal position; there is impaired resonance where it was tympanitic before, and this will become more pronounced as the fluid changes. A definite fluctuation is present, so we can state positively that fluid exists in this abdomen.

There are several rather interesting considerations, and one is that associated with the diagnosis of a cirrhosis. Can we make, or would we be justified in making, a diagnosis of cirrhosis of the liver? The history as to a specific infection is negative; she does not admit a specific infection. The use of alcohol has been rather limited and extends over only a short period of time. She does not give us the impression of an alcoholic. The signs are not those of a cirrhosis; the liver is not small, there is no collateral circulation and no enlargement of the spleen to justify us in making that diagnosis.

Then, as to some of the other possibilities: What is that tumor mass in the lower portion of the abdomen? This patient has not menstruated for three months; is she pregnant? There is a history of vomiting, but we fail to find any other presumptive signs of pregnancy. There are no changes in the breasts; the vaginal examination is negative as to pregnancy, and the tumor mass is too large for a possible three months' pregnancy.

The statement was also made that this might be a sarcoma, a carcinoma, an ovarian cyst, or a localized peritonitis, but in the presence of a localized tumor in the hypogastrium one of the first considerations is that of a distended bladder. This patient has not urinated since 8 o'clock last night and this tumor has gradually developed since that time. After the paracentesis, and on repeated examinations, this tumor was not present until this morning. A catheterization will remove this tumor entirely. It is only a distended bladder.

Now, as to the original condition: What has produced this gradual increase in the size of the abdomen; an ascites producing on paracentesis $10\frac{1}{2}$ liters of straw-colored fluid with a specific gravity of 1020 and positive cultures? We feel justified in saying that the cultures were not due to accidental infection. Every possible precaution was taken to avoid a contamination, and a separate puncture was made to obtain fluid for cultures; not the ordinary paracentesis puncture.

Vaginal smears from the patient were made and examined carefully, especially for gonococci, but were negative. The pelvic examination, when the bladder is empty, is negative. There is no involvement of the uterus, the tubes, or ovaries, and we are not justified in making the diagnosis of a tubular or ovarian involvement.

The clinical symptoms and signs justify a diagnosis of peritonitis, and the laboratory findings indicate that it is probably due to a staphylococcus.

Subsequent History.—This patient entered the hospital May 20th; was aspirated the following day. The fluid did not reaccumulate; the pulse, temperature, and respirations soon became normal. Within two weeks all symptoms and signs had disappeared, and she left the hospital June 6th.

PARTIAL OBSTRUCTION FROM A GALL-STONE IN THE COMMON DUCT (PREVIOUSLY OPERATED)

DR. TICE: What is the diagnosis?

VISITOR: There is an enteroptosis and possibly gastroptosis; she has been operated for gall-stones and still becomes jaundiced at times.

DR. TICE: Why does she become jaundiced?

VISITOR: It might be due to pressure or tension or the formation of more stones; there may be a constricting band of adhesions.

DR. TICE: This patient is twenty-five years old. She entered the hospital on the 19th of April, five weeks ago, and the history obtained at that time consisted of a postoperative discharging biliary sinus, jaundice, and a sensation of pressure or aching in the back. A feeling of malaise, of chilliness and weakness, was also present.

Her first definite hepatic colic occurred two weeks after the birth of her first child, one year ago, again emphasizing the frequent influence of pregnancy upon gall-bladder infections. This pregnancy and labor took place in the old country. A premature child was born and lived but a short time. The hepatic colic, which appeared two weeks after her confinement, definite in nature, was not accompanied with jaundice; preceding its onset there had been a feeling of discomfort in the liver area. Three months ago she gave birth to her second child, which was also premature, a seven months' pregnancy. The child lived only two weeks.

After getting up and about since her second confinement she was seized with her second definite attack of hepatic colic, and seven weeks before entering this hospital she was operated at Michael Reese Hospital on a diagnosis of cholelithiasis and some forty-five stones were removed. She was in the hospital for five weeks. Shortly after leaving the hospital she suffered from her third attack of hepatic colic. Since that time she has had several distinct attacks, and with each one of them she has become jaundiced. She complains of a feeling of tightness in her chest and discomfort in her back, with sharp stabbing pains in the region of her liver.

Her previous history, aside from that already related, is negative. There is no history of alcohol or a specific infection.

The question which now confronts us is, Why does this patient suffer from hepatic colic after having had a gall-stone operation, and why does she continue to have her jaundice? The biliary fistula at this time is closed, and has been so for the past ten days; but was discharging when admitted. There are, of course, some conditions which we must bear in mind, particularly those conditions which we have mentioned before as capable of producing

jaundice, which is quite definite, as you can observe from the external surface and also from the conjunctivæ.

Some of the possibilities which we have mentioned consist of the factors concerned in the production of the colic as well as the jaundice, associated not only with the presence of stones, but the usual accompanying inflammatory processes or the formation of adhesions. As you know, all patients with gall-stones do not necessarily have colic, nor do all the patients with jaundice at the time of operation have stones; 15 per cent. of the typical hepatic colic attacks are not associated with stones. We have here, however, the history that some forty odd stones were removed, but nevertheless this patient's jaundice and attacks of colic still continue.

In our effort to determine the condition we have, of course, resorted to test-meals, for the reason that occasionally the jaundice and attacks of colic are associated with gastric involvement, a carcinoma, peptic or duodenal ulcers, or adhesions secondary to gastric involvement. The patient has received, up to the present time, four Ewald meals, and each one, with the exception of the first, reveals only a slight increase in free hydrochloric acid, no lactic acid, and no blood. The stools are negative for blood, but positive for bile. A skiagraphic examination was made after a barium meal was given, and this shows a partial constriction of the stomach, but a constriction that is not permanent. In a very short time the barium escapes from the upper portion of the stomach into the lower portion, and, as you will observe, the stomach is very low; the lower portion extends below the crest of the ilium. A hypocondition, but not associated with any interference in the emptying nor with any actual constriction or irregularity. The gastro-intestinal examination, so far as we can determine, aside from the position of the stomach, is negative. One motor meal was given and the stomach emptied promptly within five hours.

The more serious considerations are concerned with the possible local conditions. At the time of the fluoroscopic examination it was possible to demonstrate the presence of definite adhesions, and this we can do by local examination. When we

make traction on the abdominal wall we will notice that at the point where the sinus existed there is a distinct retraction or depression of the scar. We can palpate from this scar, running downward and backward, a definite tumor mass, giving us the impression of an elongated body—probably adhesions, with the thickened, elongated gall-bladder attached to the abdominal wall—and, on deeper palpation, the lower edge of the liver is felt a couple of fingers below the costal margin. The left lobe of the liver is easily palpable. The spleen is not palpable.

The problem here deals with the existing pathology, particularly as to the presence of adhesions or a gall-stone. Is this patient suffering from a gall-stone in the common duct with partial obstruction, a stone which was not removed at the time of the operation, or are there adhesions producing a kink in the gall tract with attacks of colic, or is the trouble simply due to an existing infection, which is a marked factor in the production of hepatic colic and jaundice?

As to the influence of the infection, it is rather difficult to account for these symptoms on that basis, for the very reason that up to ten days ago this patient was suffering from a biliary fistula; the gall-bladder and ducts were being drained, but the attacks of colic persisted just the same. So we rather discredit the existence of an infection. As to the presence of a gall-stone in the common duct, which could easily have been overlooked at the time of operation, it must be remembered that this patient has only a slight degree of jaundice, not the deep, intense, almost black jaundice which results from complete obstruction. Bile is present in the stools, so the obstruction can only be a partial one. The history also states that she has suffered from chills and fever, which so frequently accompany partial obstruction of the hepatic duct, the so-called hepatic fever and chills stimulating attacks of malaria.

The other condition most likely is that of adhesions. Opposed to this assumption, however, we have the fact that no adhesions were found at the time of operation. The attacks of colic appeared before the operation and have persisted since. We know that adhesions are present between the abdominal wall and the

gall-bladder and they can easily be accounted for, but if adhesions were responsible for the pain, we would expect them to produce more frequent and persistent attacks. If the condition were due to adhesions, why should she be perfectly comfortable for several days and then all of a sudden suffer from an attack?

So far as we are able to ascertain, we believe that this condition is due to the presence of a stone in the common duct with partial obstruction. The patient has been advised to undergo a second operation. The indications are clearly surgical. It makes no difference whatever whether a stone is lodged in the common duct or whether there are adhesions, it is purely surgical, and no advice aside from surgical interference can be given. She has consented to another operation, and will be transferred to the surgical side on the tentative diagnosis of a partial obstruction from a gall-stone in the common duct.

NOTE.—Patient was transferred to surgical service May 26th. Operation was, however, postponed and she left the hospital June 8th, condition unchanged. After a few days at home, the attacks of colic became worse and she was re-admitted to the surgical service July 6th, and was operated the following day. Notes from the surgical history: "Opened gall-bladder. Separated adhesions and freed gall-bladder. Cut down over stone in common duct. Put small rubber tube in latter. Ligated cystic artery and removed gall-bladder. Rubber drains, wound closed."

Patient was discharged July 29th.

CHOLELITHIASIS: ITS ETIOLOGY AND PATHOLOGY. EARLY SURGICAL INTERFERENCE PREVENTS COMPLICATIONS

THIS patient is sixty-five years old, and entered the hospital on the 28th of April. The history obtained through an interpreter consists of a sudden attack of severe pain in the right hypochondrium, which began five weeks before her admission.

The pain has persisted since that time with intervals of relief. It has radiated around to the back and with but a slight amount of discomfort in the shoulder. Chills and fever followed the initial pain. Several chills have occurred since that time, and at such times the pain is very much worse. The patient believes the chill is followed by a fever. Vomiting also began immediately after the onset of the pain; she has vomited many times since, but never any blood. Tenderness and sensitiveness is present in the region of the liver. Two days after the attack of pain it was observed that she was definitely jaundiced. This jaundice increased in degree up to the time she entered the hospital. Her bowels have been fairly regular; there is no history of gastro-intestinal disturbance aside from the vomiting. There has been a marked diminution in her desire for food; practically an absence of appetite since her present illness. She believes that she may have lost some weight during the last few months. This, however, is not positive.

Past History.—An attack similar to the present one occurred two years ago, which lasted two weeks, the pain appearing in the right hypochondrium, severe, sharp, and colicky in nature, followed by jaundice. According to the interpreter, the pain ceased suddenly, the jaundice disappeared, and she remained well up to the time of this present attack. The family history is negative; there is no history of carcinoma or tuberculosis in the family. The menstrual history is also negative; she denies a venereal infection. The patient uses a moderate amount of beer, but has never used whisky.

On the day the patient entered the hospital the temperature was 101° F., pulse 108, respirations 22. The same afternoon, four hours later, the temperature was 100° F., pulse 106, respirations 20. The following morning at 8 o'clock the temperature was 100° F.; that afternoon, 98.8° F. The next day in the morning it was 98.2° F., in the afternoon, 98° F. Since that time the temperature has been practically normal, perhaps slightly below normal in the morning, and more nearly normal in the afternoon. The pulse has gone down to 80, 86, 88, the respirations 20 to 22.

Upon examination, we are first attracted by the uniform discoloration, and some of you will recall, when the patient was in the clinic a week ago today, this discoloration, or jaundiced condition, was very much more pronounced than it is today. At that time it was a very deep, high-grade jaundice, but even now the examination of the eyes with artificial light reveals a definite pigmentation. The mucous membrane of the mouth and the external cutaneous surface of the entire body present this same pigmented appearance.

The patient has referred practically all of her complaints to the liver, or the right hypochondrium, and as a result we will direct our attention to that region. The abdomen at this time is not distended; there is nothing abnormal except the pigmented skin. On light palpation there is no rigidity; the abdomen is perfectly soft and relaxed. On deeper palpation, we can determine the lower border of the liver, three fingers below the costal margin. There is no tenderness of the liver, at least no marked tenderness, and no discomfort until we approach the region of the right rectus or the neighborhood of the gall-bladder. Here only moderate pressure produces discomfort. No tumor mass, however, is palpable—only the border of the liver. Both the surface and the border of the liver are perfectly smooth; no marked irregularity or nodular condition. There is no tenderness or discomfort in the epigastrium. The spleen is not palpable either in the dorsal or lateral position. Percussion of the abdomen reveals nothing abnormal. The upper border of the liver is in the fourth intercostal space.

The further findings are connected with the laboratory examination. The day after the patient entered the hospital the urinalysis revealed a urine with an acid reaction, specific gravity of 1030, a small amount of albumin, a few leukocytes, but no casts. The chief finding consisted in a great increase in the bile. White blood count was 11,110. A second urinalysis two days after she entered was identical with the first. A stool examination made on the 4th of May revealed the presence of bile; no blood was found.

An Ewald test-meal was also given, and 35 c.c. recovered;

the chemical and the microscopic examinations revealed normal findings.

A diagnosis has been made of cholelithiasis or gall-stones. The history is typical: Two years ago the patient suffered from a similar attack; during an interval of two years she was practically well, and was then suddenly seized with severe pain in the hepatic region, with nausea and vomiting, with the development of jaundice, with recurring chills and probably fever. Subsequently she was brought to the hospital, and on the first day had a temperature of 101° F. About twenty-four hours later the severe pain diminished, the temperature became normal, and the jaundice has gradually faded. From these symptoms and signs we can make a very definite diagnosis of cholelithiasis.

There are some other conditions, however, which simulate cholelithiasis to which we should direct our attention, and perhaps the most likely and reasonable possibility is some gastric involvement. The examination, however, of the epigastrium reveals no suspicious findings; the test-meal was negative, as was also the stool examination. Recurring attacks of colic in the region of the liver or in the right side of the abdomen are often associated with a peptic or duodenal ulcer, but these we can also quite definitely exclude. The same is true of a kidney-stone colic; the pain was not down in the inguinal region; it was not associated with any urinary disturbance; no blood was found at any time in the urine.

Some of the more interesting points in the diagnosis of cholelithiasis are concerned with the etiology and pathology of the disease. At the present time it is quite generally accepted that there are at least two very essential etiologic factors contributing to this condition. These consist, first of all, in an infection of the gall-bladder, most frequently with the typhoid or the colon bacillus; but other organisms may be found, particularly the streptococcus in modified form, and produce identical changes with those of a colon or a typhoid bacillus; the infection perhaps is most frequently carried through the portal circulation. After the gall-bladder becomes infected a chronic cholecystitis ensues. These two circumstances combine to interfere with the function

of the gall-bladder, which results in the production of calculi. The contributing factors are in the mode of living. A quiet, sedentary life, particularly with constipation, assists in the production of a cholelithiasis. Probably the matter of diet is of some consequence; it is believed that an excessive amount of meat may have some effect. This, however, is doubtful when we consider the greater frequency of gall-stones among the Germans and Austrians than among the English, as the former consume much less meat than the latter. The use of alcohol probably has a far greater influence upon the formation of gall-stones than the use of meat or nitrogenous foods.

As to the symptoms, they are quite variable. A good classification of the symptoms of gall-stones is to group them as septic and aseptic or mechanical. Perhaps the most useful classification is to divide the symptoms into general and local, the latter being the disturbances caused by the presence of a stone or stones in the gall-bladder or in the cystic duct or the common duct.

Under the general symptoms, gastro-intestinal disturbances should be mentioned first. This is a chronic manifestation that is being more frequently appreciated than formerly. A patient with a so-called dyspepsia or indigestion and discomfort after eating, which persists over a long period of time and is not influenced by dietetic treatment, should be seriously considered as a possible gall-stone case. Then the second symptom, under the general symptomatology, is that of discomfort or pain. This can be very well subdivided into two types—the dull, heavy, boring pain or pressure-like sensation in the region of the gall-bladder, more or less continuous, perhaps intensified after the taking of food, and the severe, sharp, paroxysmal attacks more typical of gall-stones.

An interesting question is that associated with the causes of a gall-stone colic. We know from our postmortems that practically 10 per cent. of all individuals have gall-stones, but only a very small percentage ever suffer from gall-stone colic. The colic is precipitated either by mechanical causes—*i. e.*, a fall, a blow, or a jar, which incites contraction of the gall-bladder, or,

far more frequently, the peristaltic contraction of the gall-bladder is initiated by the infection which is present.

The infection is responsible for an effort on the part of the gall-bladder to relieve itself of its contents, and this forces the stone into the cystic duct and produces the colic. That the so-called gall-stone colics are always due to the presence of a stone in the biliary tract is not true, for surgeons frequently find the gall-bladder and biliary tract perfectly free, and in such instances the colic is due to the infection and not to mechanical irritation by a calculus.

As to the occurrence of jaundice, it appears in approximately two-thirds of the cases only; a little over 33 per cent. never have jaundice, which is attributable to the fact that the calculus is engaged only in the cystic duct; even if the stone passes on into the common duct, jaundice does not appear unless the stone is large enough to produce an obstruction. If the stone is extremely large and becomes engaged in the cystic duct, it is possible that jaundice may appear before the stone enters the common duct, or it may result from pressure of the glands about the common duct secondarily involved by the local infection.

The conditions are variable as to what may happen in the gall-bladder when a stone becomes engaged in the cystic duct. If the stone is impacted in the cystic duct, then after a short time the pain disappears. The gall-bladder becomes enlarged and distended, the bile is gradually absorbed, and is replaced by a mucous material. Subsequently, if a thickening of the gall-bladder occurs, a contraction may result. On the other hand, if the stone in the cystic duct does not produce a complete occlusion, then, as a rule, the accompanying infection will produce a thickening of the gall-bladder, and, particularly if other stones are present, the gall-bladder becomes contracted and shrunken upon the contained calculi.

When the stone is in the common duct the conditions depend entirely upon the size and location of the stone—whether there is a complete occlusion or only a partial one, whether the stone is finally released and escapes into the intestine. The complications and sequelæ are, of course, exceedingly variable, including

infection of the gall-bladder, infection of the liver, hepatic abscess, rupture of the gall-bladder or perforation, gangrene of the gall-bladder, fistula into the intestine, or, externally, peritonitis and enterocolitis.

In the matter of treatment, of course, it may be divided into the medical and the surgical. As to the medical treatment, perhaps, first to be considered is that concerned with the relief of the paroxysm. If the patient is observed at the height of the attack, the indication is to relieve the pain, and this can usually best be done by a single or repeated dose of morphin hypodermically. If the pain is not relieved after a reasonable amount of morphin, then chloroform perhaps is best, relieving not only the pain, but also producing a relaxation and permitting the stone either to slip back into the gall-bladder or pass on into the intestinal canal.

The question of medical treatment, with the object of dissolving the calculi, is one of interest, but not associated with many encouraging results. This question is intimately connected, of course, with the character of the calculi and the solubility of the particular constituent. The cholesterin type is soluble, and it is possible to introduce them into the normal gall-bladder of the lower animals and obtain a total disappearance of the calculi, but in cholelithiasis in the human subject the conditions are quite different. The bile is already overcharged with cholesterin and consequently fails to dissolve the calculi.

Furthermore, the presence of the calculi and the abnormal character of the bile prevent the gall-bladder from properly contracting. The normal irrigation of the gall-bladder does not occur, and, as a result, the calculi are not brought into contact with a normal or usual amount of bile. Consequently, the idea of giving any drug or substance with the object of dissolving calculi is based upon very poor experimental and clinical grounds.

The larger proportion of the so-called "cures" through the administration of solvents, especially olive oil, is based simply upon the assumption that the hard, saponified masses discovered in the stools are really calculi.

Before going into the surgical treatment we will discuss

briefly the prophylactic treatment of calculi with reference to the treatment of typhoid, colon bacillus infection, or some other type of infection. When a cholecystitis occurs, as is not infrequent, following a typhoid, or due to a colon bacillus, or intestinal infection of some other type, the essential treatment consists of drainage of the gall-bladder. A cholelithiasis is only the end-result of a cholecystitis, and if we wish to prevent the possible occurrence of a cholelithiasis then a cholecystitis should be treated surgically by drainage. When calculi have occurred and a patient has suffered from an attack or recurring attacks, then the treatment is definitely surgical. It is true that a patient may have but a single attack, pass the calculus, and never have a recurrence. Usually, however, the calculi are many and recurring attacks are the rule. This means that it should be considered a surgical case from the standpoint of the relief of the cholelithiasis, either by drainage of the gall-bladder or, best of all, an excision of the gall-bladder.

When this is done, the possibility of a not infrequent complication, the development of a carcinoma, is very much minimized. A cholelithiasis which is permitted to go on for months and for years quite frequently terminates in carcinoma; as a result of surgical interference the danger of a carcinoma is removed or minimized.

This patient entered the hospital with the symptoms and clinical findings of a cholelithiasis, but from the day she was admitted she has quite rapidly and continuously improved. Today she is entirely free from pain of any kind, except for a tenderness on pressure over the gall-bladder. The jaundice has practically disappeared; there is an abundance of bile in the stools; and bile has almost disappeared from the urine.

One of two things happened—she either passed the calculus or it slipped back into the gall-bladder. But, in consideration of the fact that she has had two definite attacks of gall-stone colic, regardless of her present condition, the advice to give and to follow is surgical interference. This patient will be transferred to the surgical side either for drainage or, better, removal of the gall-bladder.

NOTE.—May 24, 1915. The patient presented in the clinic a week ago today, on whom we made the diagnosis of a cholelithiasis and cholecystitis, suffered from another gall-stone colic that evening. She was transferred to the surgical service and operated the following day. The notes made at the time of operation are to the effect that gall-stones and cholecystitis were found; the gall-bladder was distended, containing a slightly dark, sticky fluid and five medium-sized stones, which were removed; a rubber drain was inserted and permanent drainage established. Her condition has remained very satisfactory up to the present time.

CHRONIC INTERSTITIAL NEPHRITIS. HYPERTENSION; ITS INTERPRETATION AND TREATMENT

THIS patient is seventy-two years old, and entered the hospital about three weeks ago. The history obtained was as follows: She has had rheumatism four times during the past three years. Her last attack began in August, eight months ago, and has continued ever since. The articular involvement began in the hips, later appeared in the knees, then in the back, and has remained constantly in some of the joints from the beginning. Her joints have never been very painful or tender. There is a history of a long-continued constipation. There is difficulty in retaining the urine. Appetite has not been affected. There is no loss in weight, no complaint of cough or sore throat. During the last month she has had rather severe and frequent headaches.

Past History.—She had pneumonia and typhoid years ago. Family history is negative. She has given birth to two living children and has had eight miscarriages. A venereal infection, however, is denied. There is a history of moderate use of alcohol. The temperature when she was admitted was 99° F., pulse 120, respirations 22. The following day the temperature was 98.6° F., pulse 80, respirations 20, which have persisted up to the present time.

Physical Examination.—As you will observe, the patient is well past middle life, but well nourished. The history informs us that she has lost slightly in weight, but even now her general appearance would indicate a good state of nutrition. On examining the eyes we find that the pupils are equal, slightly dilated, respond promptly to light and accommodation; a well-defined arcus senilis is present. There is no edema, no swelling or puffiness of the face nor of the eyelids. Examination of the mouth reveals the absence of a number of teeth; the gums are red and inflamed along the borders; pressure on the gums very readily produces bleeding, and it is possible to express some pus from around the teeth—a moderate grade of pyorrhea.

The chest, upon inspection, reveals nothing abnormal, with the exception of possibly a relative increase in the anteroposterior diameter. The apex-beat, on inspection, is in the fifth intercostal space, in the nipple line. Percussion over the lungs anteriorly reveals a slight hyperresonance. The liver dulness is at the fifth intercostal space. The right border of the cardiac dulness one finger to the right of the sternum, the base at the second intercostal space, the left border midway between the nipple and the anterior axillary line.

On auscultation over the lungs expiration is relatively prolonged. Over the heart, at the apex, the two tones are present, the first associated with a definite systolic murmur; the second pulmonic is accentuated; over the aortic area no murmurs are present, but the second aortic sound is loud and ringing.

Examination of the chest posteriorly reveals nothing abnormal on inspection; on percussion a slight hyperresonance, and on auscultation practically the same findings as indicated anteriorly. Over the lumbar and sacral region no edema is present. Examination of the abdomen, aside from the adiposity, is entirely negative. The liver and spleen are not palpable.

Upon inspection of the lower extremities we are at once attracted by the rather unusual shape. The feet and ankles are apparently normal or, at least, relatively small. There is no swelling and particularly no edema of the feet or the lower two-thirds of the legs. The swelling appears in the upper third of the

right leg and approximately at the middle of the left leg. Above this point there is very definite edema, distinct pitting, as you can observe. This condition extends above the knees and involves the thighs. The patellar and tendo achillis reflexes are present. As to the nature of this condition we will return later.

The morning following the day on which this patient entered the hospital the urinalysis revealed a specific gravity of 1016, the presence of albumin, a large number of hyaline and a few granular casts. Her blood-pressure is 250 systolic and 140 diastolic.

As related, we have some very definite clinical findings on which we can make at least a tentative or partial diagnosis. The presence of albumin, hyaline and granular casts, an increased blood-pressure, the systolic 250, aside from other physical signs, of course are strongly indicative of a chronic interstitial nephritis.

In the examination of the heart we have already referred to the increase in the cardiac dulness to the left, associated with a systolic murmur at the apex, and over the base of the heart a loud, ringing second aortic. These are the findings, so far as the heart is concerned, of a chronic interstitial nephritis, with an increased blood-pressure. Examination of the arteries show them to be hard and sclerotic, definitely beaded. The radial, temporal, and carotid arteries are hard and sclerotic.

Perhaps the most interesting feature associated with this patient is the high blood-pressure—250. We have another patient in the ward, a young lady thirty-two years old, with a chronic interstitial nephritis, and a blood-pressure this morning of 260. The subject, as you know, has received an enormous amount of attention during recent years, and some rather definite conclusions have been obtained, particularly with reference to the significance of an increased blood-pressure in relation to the prognosis, and then our almost complete failure in our therapeutic efforts to control the degree of blood-pressure to any appreciable extent.

It is quite generally accepted at the present time that a systolic blood-pressure not exceeding 160 and a diastolic of 100 are within

the normal limits. It is also generally admitted that the diastolic blood-pressure is of greater value than the systolic. The systolic may vary, and is quite easily influenced by a large number of factors, while the diastolic is not. A diastolic of 100 or above is usually taken to indicate a hypertension. In the clinical consideration we must make a distinction between the so-called simple hypertension and the organic hypertension; the simple, non-organic hypertension is not associated with any demonstrable lesion in the kidneys, vessels, or heart.

As to the etiologic factors producing a hypertension, that, of course, is another question. The tendency at the present time is very decidedly in favor of an independent etiologic relationship between arteriosclerosis and hypertension. Formerly it was quite generally believed that involvement of the vessels in the form of an arteriosclerosis preceded hypertension. Now the belief prevails, and is becoming general, that the hypertension, and particularly the long-continued hypertension, antedates any anatomic changes in the vessels or even in the kidneys or the heart.

Of the pathologic hypertensions, that associated with a nephritis is of the greatest importance, and it must be understood that hypertension is not associated with all forms of renal pathology. The most frequent anatomic change accompanied by hypertension is a chronic nephritis. When we observe a pronounced increase in the blood-pressure it is usually accepted as indicating a chronic interstitial nephritis.

This brings up a very interesting problem and one which is by no means settled—*i. e.*, the determination of the cause of hypertension and especially its secondary effects on the heart in the form of cardiac hypertrophy. That the heart increases, becomes hypertrophied, and that mitral insufficiency and other lesions develop is common knowledge and was accurately demonstrated by Bright, who first described the disease which now bears his name. It was Bright's idea that the cause of the cardiac hypertrophy was the retention of toxic material in the blood, and, second, that there was a contraction of the peripheral circulation, increasing the work of the heart.

In explanation of the cardiac hypertrophy, or the cardiac involvement associated with a chronic nephritis and hypertension, we may refer to the so-called mechanical theory which was first described by Traube. In chronic interstitial nephritis there is a decrease in the size of the kidneys, and in order to maintain a sufficient secretion it was evidently necessary for the heart to hypertrophy so as to increase the flow and force of the blood in the renal circulation. This mechanical theory of Traube for a time was somewhat disregarded, but recently has again been emphasized. However, experimental work seems to throw a great deal of doubt upon its value and importance. Müller and Mass experimentally produced paraffin embolization of the kidneys without any effect upon the blood-pressure. Loeb somewhat modified the so-called mechanical theory, particularly in regard to the compensatory efforts of the splanchnic area, which he believed to be a secondary result of a glomerular involvement. This, however, is very doubtful, for in amyloid degeneration, where the glomeruli are chiefly involved, the blood-pressure is normal or subnormal.

The second explanation is founded on the so-called toxic theories: First, insufficient renal elimination. This has recently received the attention of Paessler and Heineke, who removed one entire kidney of a dog, and then by subsequent operations gradually removed the remaining kidney, taking a section at a time, and, so long as not less than two-thirds of the second kidney remained, no material effect was produced upon the blood-pressure. It would seem, however, that other factors may be concerned, for it is quite impossible to undertake such extensive and repeated surgical operations without implicating other organs or functions, such as the suprarenals, and probably also the nervous system. Therefore the conclusions are to be considered only of relative value.

Second, the disturbances of the internal secretions. Perhaps first to be mentioned is the production of rennin, due to destruction of kidney substance, which is absorbed and increases the tension. That this has any influence in effecting a high blood-pressure in nephritis can hardly be expected. The kidneys are small and

contracted, and that any autolysis occurs in them is very improbable.

Next we shall consider the disturbance of the suprarenals, which was first suggested by Neusser, of Vienna, and has since been amplified by the French clinicians. This, however, is quite doubtful, for the very reason that the suprarenals functionate only periodically or temporarily. In the blood of patients with hypertension it is not possible to demonstrate an excess of suprarenal extract. The impossibility of detecting an increase in suprarenal extract in the circulating blood of patients with a hypertension, as well as the absence of a hyperglycemia or a glycosuria in these patients, throws a very great doubt upon this theory.

We know experimentally that we can inject suprarenal extract and produce marked hypertension, which is almost constantly associated with an increased amount of sugar in the blood, but these conditions do not exist with the hypertension connected with chronic interstitial nephritis. Consequently, the suprarenal is not to be held accountable for hypertension.

Third, that toxic substances are set free by the diseased kidney. Just what toxic substances may be set free has not been accurately determined.

When we come back to the question of why the heart is hypertrophied in a chronic interstitial nephritis with an increased blood-pressure we are still in doubt. We cannot at this time state that one factor or another is positively the cause, and perhaps the best we can do is to ascribe the condition to a combination of factors. Not until we know what produces a primary hypertension unassociated with any organic involvement, and not until we know the substance or substances producing a hypertension, can we make any definite statement as to the production of a cardiac hypertrophy associated with a chronic nephritis.

In fact, at the present time, our knowledge in reference to nephritis is being so much disturbed and revolutionized that we are not capable of making a very intelligent classification of the various kidney lesions.

As to the complications associated with hypertension, we can perhaps best subdivide them into three groups: A cardiovascular group, in which the cardiovascular manifestations predominate, such as dyspnea, palpitation, etc., or with angina, or a rupture of a vessel, as occurs in a cerebral hemorrhage.

A second group, in which the renal manifestations predominate, where there is interference with the function of the kidneys, with uremic manifestations, a definite coma, or convulsions.

A third group, where the two are combined, and we have renal and cardiac manifestations, sometimes one predominating, sometimes the other.

From the standpoint of prognosis this, of course, varies. Not all instances of high blood-pressure necessarily mean an unfavorable outcome. A few years ago, when we first were able to determine the blood-pressure clinically, we were alarmed at the marked increase in pressure, believing that some untoward result would follow, either renal, cardiac, or vascular. Today, however, we do not look upon high blood-pressure, except when unusually high, as of much consequence. The condition is a purely physiologic one, and high blood-pressure is necessary and essential for the proper function of the kidneys. A most important feature about a high blood-pressure is the fact that we cannot modify it, at least not more than temporarily, to any material degree, even by our utmost efforts. By one means or another we may temporarily reduce the blood-pressure, but it does not remain so; it very soon returns to its former level, simply for the reason that it is a physiologic process.

In very high blood-pressure (and we shall refer to pressures above 190 as very high) the conditions are different. We may at any time encounter unfavorable cardiac, renal, or vascular complications due to the strain upon the heart and the vessels, or because of interference with the function of the kidneys.

In reference to the matter of treatment, which is the all-important consideration, we might refer to the work of the heart. According to Pope, when the heart beats at the rate of seventy times a minute, that is, 4200 times an hour, 100,800 times a day, and 36,792,000 times annually, which means, pumping $2\frac{1}{2}$ ounces

of blood at each contraction, an average of 175 ounces of blood a minute, 570 pounds an hour or $7\frac{1}{2}$ tons a day, which is equivalent to lifting 1 ton 122 feet high. You can perhaps form some conception of the work accomplished by the heart when we estimate it in pounds. This is equivalent to 10 pounds a minute under normal conditions. Any increase in the blood-pressure above normal must increase the work of the heart proportionately.

In the treatment of hypertension it must, of course, be first remembered that we have two fundamental factors. First of all, that the hypertension is an attempt on the part of nature to correct an abnormal process in the kidney; and second, we have certain factors, usually dietetic habits, mode of living, etc., which may augment the arterial tension.

It is principally with the second group of factors that we are concerned, and in the treatment of a hypertension the one factor of first importance, when the blood-pressure reaches 190, is to place the patient at rest. The patient should discontinue business and preferably be in bed, with the idea of lowering the pulse-rate and diminishing the work of the heart. If the heart-rate is increased one-half the normal, then that increases the strain on the heart by proportionately one-half.

Second, as to the diet. Usually in these hypertensions there is error in the diet, particularly in excessive use of nitrogenous foods, and frequently an unreasonable amount is consumed. In an unusually high blood-pressure the patient will do best upon a milk diet or upon a liquid diet with all nitrogenous foods restricted. Free elimination by means of the bowels will assist materially in ridding the patient of toxins or toxic material absorbed from the intestines, and will also relieve the work of the kidneys by increasing the elimination. Fluids should be restricted, particularly in cases associated with an edema. A salt-free diet, particularly if the patient has been accustomed to using salt liberally, may have a beneficial influence. Occasionally, however, the moderate use of sodium chlorid will produce a better effect upon the edema than an absolutely salt-free diet. Alcohol is not to be permitted in any form.

One of the best and most efficient means at our command in the treatment or control of hypertension is the use of hydrotherapy. Various forms of baths may be used, but the one to be preferred is the luke-warm bath. The hot baths or the cold baths influence the hypertension unfavorably. The luke-warm baths will produce the best results. Sweats, where the cardiac condition will permit, with free perspiration, will very favorably influence the high tension, but, unfortunately, in most of the cases the heart is already involved and alcohol sweats or hot-air baths cannot be tolerated. Electric baths, as well as high-frequency treatment and the radio-active baths, all have been used with varying reports, perhaps, on the whole, not favorable.

Where there is very high tension, and where there is danger of cardiac failure, or where there is danger of rupture, then a venesection should be done, which will certainly relieve the condition for the time being, though it is not permanent. The same thing may often be accomplished, particularly during a uremic convulsion, by a lumbar puncture, which relieves the intracranial pressure—practically a decompression by lumbar puncture.

This patient, in addition to the findings indicated, presents an interesting picture of an edema in the upper portion of the legs and the lower portion of the thighs. What produces that? With a cardiorenal-vascular involvement we would expect edema in the ankles or the feet. These feet and ankles are not edematous, but the edema is in the upper portion. This, evidently, is not due to position; the patient rests in bed perhaps more than half the time in the position in which you see her now, in the dorsal position, with the knees drawn up, and if the position is a factor, then the edema should appear in the legs or more dependent parts. Her knees are above the level of the heart. Has anyone an explanation?

VISITOR: It may be due to some change in the blood-vessels.

DR. TICE: Yes; when we examined the vessels in the lower extremities we found a thrombosed condition in the veins extending up to the middle of the legs. Not only are these vessels hard, sclerotic, completely thrombosed, but at certain points

hard masses are present. This condition, the obliteration by a thrombophlebitis associated with a sclerotic condition of the skin and the subcutaneous tissues, prevents the occurrence of an edema.

On account of these findings in the limbs and because of pain in the knees skiagraphs were taken, and the *x*-ray department reports that the joints show definite skiagraphic indications of an old arthritis.

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL
FOR CHILDREN)

HYSTERIA IN CHILDREN

THE girl whom we present this morning is eleven years old. She was admitted to the hospital because she was suffering from so-called "nervous attacks." Now this child was always well until five months ago, when the attacks first came on at night. She has had five similar attacks since. As near as we can learn from her parents, the attack comes on suddenly and quietly, the child becomes very rigid, particularly the extremities; she froths at the mouth and the mouth is drawn to one side. She swings her arms in all directions. This lasts about fifteen minutes, and the child awakens with a start and complains of a choking in her throat and is apparently oblivious to all that goes on about her, though the mother affirms she is not altogether conscious of the attack.

When we come to examine her we find that she is a large, normal child, of good complexion; her eyes are normal in every way, her expression is sad.

DR. ABT (to Patient): Are you sad this morning, Esther?

Patient smiles.

DR. ABT: She does not appear so sad. (After a few questions about her home and school life, which she answers well, she appears to be a bright child.) We note that she spells words well, she reads very fluently, she can do rather complex problems in arithmetic; because of her modesty she refuses to admit, though nevertheless we are led to believe, that she is one of the brightest pupils in her class.

DR. ABT (to Patient): When was your last attack?

PATIENT: Two weeks ago when it was thundering.

DR. ABT: The patient insists she has had only two attacks. She says that they occur at night and usually when she is frightened. She never has had an attack at school; indeed, she has never had an attack during the day. She does not have any premonition that the attacks are about to occur. She does not suffer from headaches.

Examining her further, we find that there is no rigidity of the neck; no asymmetry of the face, no deviation of the tongue. She has a well-formed thorax. Physical examination of the heart and lungs is negative. Examination of the abdomen is entirely negative. The right foot shows webbing of the first and second phalanges; the patellar reflexes are considerably exaggerated. The urine shows no abnormalities. Examination of the blood shows 75 per cent. hemoglobin. Otherwise the conditions are normal.

The von Pirquet test is negative. The electric irritability shows C. A. C. to be 8 milliamperes, indicating no increased nervous or muscular irritability.

We find, too, that on careful testing sensation is normal and equal in all parts of the body.

We may observe that there is nothing in the family history that will throw any light on the case.

In reviewing the findings we note that the patient has peculiar attacks which follow fright, that the movements of the extremities are inco-ordinate, more wildly throwing than convulsive or spasmodic, that she is not completely unconscious, that she does not wet her bed or bite her tongue during the attack.

We believe this patient to be suffering from hysteria. One would naturally consider whether these seizures were epileptic in character. We believe that epilepsy may be excluded because the seizures are not characteristic. We are not given any account of the rolling or turning of the eyes. There was no twitching of the facial muscles, nor were there muscular contractions of the extremities such as occur in true eclampsia, and, above all else, the child was not completely unconscious.

As a rule, severe convulsions, hysteric in character, do not

occur in children, though one does not infrequently observe twitchings in the extremities. At times, however, differentiation between epileptic and hysteric attacks must be made. In general, the rule holds that the younger the child the less likely is the condition to be hysteria and the more likely is it to be epilepsy. In making the diagnosis one must consider the environment. Is the mother or are both parents neuropathic? If attacks occur only rarely during the course of years, one would be inclined to think more of epilepsy. On the other hand, if the attacks occur in young girls near the age of puberty, if they are well marked or exaggerated, a diagnosis of hysteria is more probable. As a rule, one may say that convulsions occurring in very young infants and in very young children are most likely epileptic or spasmophilic.

Hysteria in children has much to do with hereditary conditions. In most cases if we can obtain an intimate insight into family conditions we will find that one or more of the immediate forbears are extremely nervous. This is the predisposing factor. The immediate or exciting cause is usually external; it consists of psychic or physical trauma. Children who have received corporal punishment, or those who have met with an accident involving shock or fright may show hysteric symptoms. Children who have suffered from fright from any cause, or those who have witnessed a tragedy or disaster, or those who are told horrible tales, or who read stories of suffering or cruelty or of shocking conditions may be shocked psychically. It has been pointed out that, as puberty approaches, hysteria becomes more frequent in girls than in boys, though in younger years the cases occur quite as frequently in one sex as in the other.

Definite attacks of hysteria are scarcely ever observed before the third year. Sometimes children become hysteric after they have suffered from some bodily disease. For instance, if the child has had an arthritis or painful extremities, or if an arm or leg has been in a cast for some time, contractures or hysteric affections of the limb may persist as a result of suggestion.

It has been pointed out, and I wish to emphasize it this morning, that the clinical manifestations of hysteria in children

are, for the most part, monosymptomatic. Now this does not imply that there may not be several clinical manifestations, but it does mean that, as a rule, one symptom preponderates and outweighs all the others.

This fact may be explained on the basis of the simplicity of the mental processes, particularly the imagination of children. The processes are not so complex and manifold as in adult life. The child may suffer from hysteric paralysis of arm or leg or from hysteric blindness or deafness, but he will seldom have motor and sensory disturbances associated. It has been suggested that in adults the multiplicity of symptoms may be accounted for by the frequent examination and suggestion of physicians or even friends. Hysteric stigmata, such as are observed in adults, are, for the most part, absent in children. Painful pressure points and disturbances of sensation are rare.

The disturbances of motility which are observed in the extremities are characterized by the occurrence of contractures, flaccid paralysis, and referred back to some trauma that has occurred. A fall on the back may give rise to fear of inability to control muscular co-ordination. The cause is not always physical. Sometimes a psychic condition is the cause. These children may be ataxic. On attempting to walk they toss and tumble from side to side and fall, but on being put to bed they move and toss their limbs in every direction.

Choreiform movements, *tics*, and *blepharospasm* are not uncommon hysteric manifestations in children. The movements sometimes occur in hands and feet, and, while not always typically choreiform in character, it is always difficult to differentiate the movements from those of true chorea. In hysteria, if the attention is arrested the movements may cease entirely for some time. In hysteria the endocardial complications are absent. We usually find that the chorea occurs in children who have been restless, mercurial, and moody, and most frequently in those who have inherited a neuropathic constitution.

Hysteric chorea in its motor form resembles the infectious variety. In hysteric chorea the choreiform movements are more likely to be confined to individual muscle groups, few in number,

while in the infectious type many groups are involved, thus constituting the general muscular twitching. Hysteric chorea may occur in epidemics. Entire classes in a girls' school sometimes show this disorder, and in such cases imitation as a cause of chorea is strikingly brought out. A child who has previously suffered from chorea, which may have been a genuine Sydenham variety, may subsequently suffer from a hysteric form if subjected to great irritation or psychic insult.

I desire to tell you about a twelve-year-old girl that I was called in to see last evening. The patient is one of five children. The family are intellectuals. The father is a college professor; he is pre-eminent in his department of learning. The mother is a very gifted woman; she has read and traveled and mingled with the world. She is quick, keen, perfectly stable, though very alert. She stimulates thought in conversation.

The patient is tall and thin, quiet under natural conditions, has read more than most girls of her years, writes poetry which is considered to be of good quality, philosophizes about the world and people in a way that is far beyond the ordinary child of her years. The mother says she is not emotional, that if she is given to fears or emotions they are unknown to her; at least she represses them. The mother states that she is a child of wonderful self-control. She is very considerate of the feelings of others and very modest about her own abilities. The child has been brought up, the mother says, under the most approved methods. She has not been overstimulated, she has not been pushed at school, though she has made two or three grades when the ordinary child has made one. The mother admits that when she brought the poems to her she approved of them, dwelt on her intelligent philosophy of life, and the nobility of thought and the perfection of meter and rhyme. The mother did not think that this was stimulation. She thought it was simple justice and appreciation. During the last summer the family have been on a farm in the country. Our patient, the twelve-year-old girl, assumed charge of her six-year-old brother. He was full of life, very mischievous, very likely to put himself in dangerous positions from which he was at times difficult to extricate, always disregarded his own

safety in the presence of the cows and the horses or the farming implements or on the water craft. This wore on the little girl very much; she took her charge very seriously, and by the end of the summer was very much fatigued. Two weeks ago she was attacked, she says, by six bees. She was stung in several places, rushed into the house, and one of the bees was still clinging to her when her clothing was removed. She became extremely nervous; indeed, her mother says she was wildly hysteric. She suffered slightly for several days from the bee stings, slept restlessly at night, had disturbing dreams, though she did not complain, and endeavored to repress her feelings and emotions. Nevertheless, she was fretful and petulant during the day. She lost her appetite, grew somewhat thin, and several days after the bee episode she had a sudden outbreak at the supper table. Her face twitched, she made grimaces; she thrust out her tongue and complained of a feeling of numbness in that organ. The twitching gradually extended to hands and feet, and when I examined her last evening she showed undoubted choreiform movements in face, hands, and feet, marked mental irritability, characterized by spells of crying and moderate laughing. The movements were not constant. They increased when her attention was directed toward them. She had no fever and no cardiac involvement. I considered that this was a case of hysteric chorea in a neuropathic and fatigued child, casually related to the bee stings.

Henoch described a group of hysteric choreas under the name of "chorea electrica." These patients manifest their disorder by short, lightning-like twitchings, particularly in the muscles of the shoulder, neck, and trunk. I do not remember ever having seen a case which sufficiently complied with the descriptions of Henoch so as to include it in this class. Mild hysteric manifestations, such as twitching of the mouth and extremities, particularly the arms, hysteric laughing, crying, and coughing, are not uncommon in children.

Sometimes hysteria expresses itself as an aphonia; in some cases there is a complete loss of speech, though at times they can speak in whispers. In some cases loss of voice is associated with loss of motion of the lips and tongue.

Hysteric gastric disturbances express themselves as anorexia and dysphagia. You will remember that we discussed anorexia in a previous clinic.

Hysteric headaches most often occur when it is most suitable for the child, and disappear when most convenient. The headache usually occurs when the child desires to remain home from school, or if he has been in mischief and desires to avoid punishment, though if the child is to participate in some pleasurable recreation the headache is postponed or disappears shortly. Similarly, neuralgias may occur. This condition may take place in or near a joint or in some part of the body that has been subject to trauma or after a blow, or a push or a fall. Sometimes the patient may complain of a pain in the scar of a healed wound, especially if the overanxious parents are directing solicitous inquiries concerning the presence of pain or discomfort.

Visual disturbances are not common, though children who have had conjunctivitis or keratitis frequently complain of photophobia. Sometimes they complain of spots before the eyes. Hysteric blindness is not common, though I recall a case in my own experience where a three-year-old child simulated blindness—much to the terror of the family group. As is usual in these cases, she was the only child with a very neuropathic heredity, and had shown, even at this age, various signs of nervous instability. Early one morning, after having been reprimanded by her mother, she complained of sudden blindness and pain in both eyes. Upon arriving at the house I found that the terror-stricken family were grouped in a darkened room, all weeping and gesticulating, and the child was crying that she was blind. The pandemonium was indescribable. After clearing the room of every member of the family except the child and holding her before the bright daylight I observed that her pupils responded. I assured her that she could see, and very soon she and I were discussing the sights on the streets below. Her vision was restored.

Disturbances of hearing are not very common. I remember a little patient who presented every possible manifestation of hysteria at one time or another and who at times simulated symptoms of deafness. Strange to say, she always heard the disagree-

able things which were said about her condition when physician and nurse were discussing her case.

Sleep disturbances of all varieties are observed. One sometimes sees children who cannot fall asleep. Sometimes they sit up in bed, play, and refuse to fall asleep. At other times they simply remain awake or they throw themselves about and keep everybody else awake and disturb the whole household. Sometimes these hysteric children who have thrown themselves about violently, and have undergone great muscular exertion, will show a slight increase of temperature.

Sometimes children have sleep disturbances as a result of fear, and this may be associated with other hysteric manifestations. Very frequently these children refuse to sleep in the dark, and mother or nurse must sit at the bedside and hold the child's hands.

Pavor nocturnus may be considered as a hysteric disturbance of sleep. Such a child was brought to me during the last few days. Let me tell you the story of her illness and you will be able to draw your own conclusions. This little girl is six years and five months old. She is an only child. She has always been very nervous. She has had no sickness except intestinal trouble. Last winter her little friend and playmate died, which made a deep impression on the patient. The little patient is very precocious, of an inquiring mind, and particularly quick at repartee. The mother is a self-centered neuropath. On account of her infirmities in this direction the mother is not a pleasant person to meet. I might say more, though it would be along the same line. The mother's family are all neuropathic. The child has received violent attention from the mother and maternal grandmother. The child's father is a normal, sensible business man. A short time ago they took the child to a lake resort and there she developed nervousness. On the auto ride to the lake the child was terrified by a herd of cows and became possessed of fear and grew demonstrative. One day she heard some discussion about Platt's Chloride as a disinfectant. She misunderstood the discussion and thought that chloroform was referred to. She found out from one of the neurotic elders in the family, upon inquiry, that

chloroform was used in killing dogs. After obtaining this information she became violently hysteric; she made many gyrations with her extremities and body, and screamed that she did not want to die. A little later she overheard a conversation between two nurses in which one nurse related that she "had a horrible dream last night," had been walking in her sleep and she was so frightened that she thought her heart had stopped beating. That night the child went to sleep as usual, but about 4 o'clock in the morning she awakened screaming and crying violently. The refrain was that she did not want to die. She roused all of the guests, though in twenty minutes she was asleep again and did not waken until the rising hour. The next day she seemed normal; her attack was not referred to, though she did say that she feared cows, chickens, and engines. In two nights a similar attack occurred. During the day she went in bathing and was fond of it, but she refused to ride in a small boat. During one of the attacks her mother whipped her without effect. The attacks occur nightly. A short time ago she was brought in from the lake. She had one attack on the train and another on the street car. She had an attack last night, and after crying that she was going to die her mother informed her that she was not. She answered, "But my friend, Hazel, died." At present the mother will not permit herself to be separated from her child. She refuses to isolate the patient and the attacks occur nightly. I might say that we made a thorough physical examination, including examination of the urine and blood, and the physical condition is normal. I believe after hearing this history there will be no disagreement on the diagnosis of hysteric sleep disturbance.

The psychic stigmata make themselves evident. The children are not infrequently untruthful; they want to be seen, heard, and to receive marked attention; they easily feel themselves injured in their pride; they feel that they are treated unfairly or that they are not receiving due recognition. They are very moody, disturb other children in their play, are insulted if they cannot have the toys of other children, and pout and sulk when they cannot have their own way in the play or the game. These

children are not exactly mentally defective, though it should not be denied that in most hysteric individuals there is a screw more or less loose. At least one may say that they are not in a condition of mental equilibrium.

Perversions of sex development are sometimes indicative of hysteric stigmata, and hysteric children are more likely to masturbate than normal children.

Prognosis.—In the monosymptomatic variety the prognosis is good, though where hereditary degeneracy exists, or where numerous hysteric stigmata occur early in life and where the environment is tense and everyone is nervous, it is doubtful whether the hysteria will be arrested or cured.

Treatment.—In families where the parents are nervous, physicians should insist upon a rational training and discipline. The child should be placed in surroundings where there is quiet, order, and serenity. If the parents are nervous they should learn to control themselves, but the difficulty is that if they could control themselves they would not be nervous. It cannot be emphasized too strongly that before the child is pronounced hysteric it should be carefully examined, so as not to overlook some bodily or some other nervous disease.

Children who are extremely nervous should be removed to a school or some institution where pedagogic methods are employed and discipline exacted, and where suitable bodily exercise is a part of the daily routine.

Suggestive therapy is most important, though hypnotism, as a rule, should not be resorted to. Sometimes mere isolation or removal from the family effects a cure. Particularly should visits from the mother and the old nurse be interdicted. Isolation with ordinary suggestion and persuasion may be sufficient. Some form of electric apparatus may be used to reinforce the suggestive measures. The galvanic or faradic or high-frequency current may bring relief. The patient's ailments should not be discussed in his presence except that he be reassured that progress is being made.

According to Bruns, the method of surprise should be employed. For instance, a child with aphonia may be shown that

it is possible to talk. Strong faradic current may be passed through the larynx with the positive suggestion that upon the application of this method of treatment speech will return. It nearly always does in one or two treatments. Or where a paralysis occurs, forcible bending or electricity, associated with command, may effect a cure. In the same way, those who suffer from astasia-abasia are assisted to stand and to walk. Very often hydrotherapy acts as a powerful suggestion. For instance, they may be put into a warm bath followed by a shower of cold water.

The treatment should not be too severe at first, and should not tend to frighten the patient. These patients should be treated with utter disregard.

Hysteric patients desire to be noticed. They desire to impress their importance on those who surround them. They desire a large modicum of sympathy. One should pay no attention to the patient, and should apparently show no concern for the symptoms. If this is done, the complaints frequently disappear. The child may be placed in a room, curtains drawn, little attention paid to him, except to say that he will be cured in a short time and that his toys will be returned to him as soon as the paralysis or other hysteric symptoms have disappeared. Drugs have been suggested, though they are of no real value unless they act as suggestants.

ENURESIS

THIS little boy, aged six years, is brought to the hospital from the Orphan's Home because of nightly bed wetting. It is reported to us that he appears to be normal in every other respect. The family history is not known.

The examination of the patient shows no physical abnormalities; his stools are normal, free from worms, and microscopic examination has shown the absence of ova. The urine is normal; the blood shows no abnormality.

The nurse who brought him into the hospital says that the patient has been in the home for two years and that he has wet the bed nearly every night since his admission; he occasionally has an accident during the daytime. The condition is becoming intolerable, and the authorities at the institution want him to remain in the hospital until he is cured.

Under normal circumstances the infant learns to control his bladder function by the twelfth or eighteenth month; babies who are less well trained, by the close of the second year.

Enuresis nocturna is a condition in which the infant does not retain his urine during sleep at the age when the sphincter muscle, of the bladder should be functioning. The same condition may be present during the daytime, and then it is spoken of as enuresis diurna. Many children wet the bed during the first sleeping hours; others late in the night, when the bladder has become distended. In some cases the involuntary urination may have a definite pathologic basis, or at least a sufficient and ascertainable cause. Children who eliminate excessive quantities of urine, and who may be said to suffer from polyuria, will frequently wet the bed at night.

Patients who drink an excessive quantity of water or fluids will, as a rule, pass large quantities of urine. I recall a little patient who had an abnormal desire for fluids—one might speak of him as a water tippler. This condition started before he was one year old and continued until I last saw him, when he was seven years old. He would cry frantically until he was given water; his thirst was difficult to satisfy; he really suffered if he was deprived of water; he desired it more than food, it satisfied him more. He would awaken several times at night and demand large drafts of cold water. His urine was normal upon examination, though he passed huge quantities and often involuntarily. No cause for this condition was ever found. The child was small in stature, very bright and responsive, but extremely nervous.

Children who have suffered from edema, either due to a kidney lesion or a nutritional disturbance, will pass large quantities of urine as the edema is disappearing. The same condition of polyuria with enuresis is observed in infants with glycosuria or

diabetes mellitus, and I may remind you in passing that occasionally a child of two or three years may fall ill with diabetes. One should be on the lookout for these cases even if they are of rare occurrence.

During the convalescence from acute diseases great quantities of urine are sometimes passed. This is true of pneumonia, typhoid, and the acute exanthematous disorders. Such children often lose voluntary control of the bladder until normal conditions are re-established.

The administration of diuretics and irritant drugs may not only increase the quantity, but also the frequency of urination, and may, indeed, lead to loss of voluntary control of the bladder. One must not forget that cystitis, bacilluria, pyelitis, vesical calculus, or, indeed, a disease of the kidney itself may lead to increased quantity or frequency of micturition, and enuresis may result. Chronic interstitial nephritis with polyuria, though not frequent in the young, nevertheless does occur and must be borne in mind.

An alkaline urine, an excessively acid urine, or a urine containing pathologic quantities of amorphous or crystalline substances may produce urinary incontinence. Malformation of the bladder or urethra may produce a loss of vesical control.

In cases of spina bifida urinary incontinence is an associated condition and arises from the cord lesion. In the recent literature attention is directed to the relation of spina bifida occulta to enuresis. Spina bifida occulta results from defective closure of the lowest portion of the spinal canal, and, in addition to urinary incontinence, deformities of the feet are observed, particularly flat feet, disturbances of sensation, and abnormal reflexes. It goes without saying that children with compression myelitis from tuberculous spondylitis, those suffering from cerebral lesions, and more particularly those who are mentally defective, find continuous urinary control difficult or impossible.

Patients who suffer from nocturnal epilepsy frequently wet the bed during or immediately after an eclamptic attack.

Very often these children are circumcised without any effect; I almost said without sufficient indication. Within the past few

days a baby was brought to me by its unsophisticated parents; it was a sight to behold—a more hopeless idiot never breathed the air of this city. He could not sit, nor walk, nor stand, though he was one year old; he was absolutely blind, he made a weird sound that resembled the cry of a loon in a quiet forest. The parents told me that they had consulted all of the physicians in their neighborhood, and among others a surgeon who proposed circumcision for enuresis. This suggestion was acted upon. They brought him in to me to see whether anything further could be done. I did not find anything more to do. There are times when our sins of commission are greater than those of omission.

Various minor causes are frequently held responsible for enuresis in children. Phimosis is assigned an important rôle in the field of general practice, though I doubt very much whether circumcision is curative. I believe that when this operation cures the disorder it acts as a powerful mental suggestion. Adenoids and tonsils have been removed by the basketful to cure the condition. I believe that there is no evidence to support the view that enlarged adenoids or tonsils produce enuresis. The operation, if successful, probably acts similarly to circumcision.

If worms are discovered, particularly oxyuris, their treatment and removal is indicated under any circumstance. If anal fistula is present it should be appropriately treated.

It should be emphasized at this point that in every case the urine should be carefully analyzed and the patient submitted to a careful physiologic examination.

It is my own experience that in the great majority of cases the urine is found to be normal and that the physical examination does not show any abnormality. The condition occurs, as a rule, in badly trained children or in those who have to a greater or less degree a neuropathic constitution, and occasionally in children who are physically undeveloped or suffering from chronic disease.

In the majority of cases children with enuresis are not otherwise ill, they present no symptoms of pain or distress, or of loss of strength; indeed, very often they are robust and strong. The mother or nurse, on the other hand, is greatly distressed. She objects to the soiled bedding, the offensive odor of the room, the

increased labor in the laundry, and she is in constant fear lest the object of her affection should sicken from the cold and moisture of his bed and clothing. She picks him up before she retires so that he may void urine; he refuses at that time, she returns him to the bed, and in a short time he drenches it. He prefers to do it in that way.

Prognosis.—The vast majority of these patients recover, some earlier, some later, a few as the result of treatment. Many of them persist after the treatment has been stopped, but eventually the condition is overcome.

Treatment.—By this time we have carefully studied the condition of the patient, and if no demonstrable pathology is found we may proceed to treat the enuresis as an expression of a functional disorder.

The patient should be placed under the best possible hygienic conditions. The diet should receive particular attention—condiments, rhubarb, horseradish, acid substances, and an excessive quantity of meat should be eliminated. Supper should be served with little or no fluid; indeed, it is a good plan to give no fluid after 4 P. M.

Further than this, the treatment should be directed along psychotherapeutic lines. In the cases of neuropathic origin suggestion is a valuable agent. One must not countenance corporal punishment by the parents, as this plan would fail more often than it would succeed, though the pioneer and master, Henoeh, tells in his lectures how he treated the cases in his clinic by a hypodermic of water and several vigorous spanks on the buttocks with almost unfailing success.

I have satisfied myself that the attitude of the parents toward these patients is at times wrong; they are impressed that the child has a serious and protracted disorder, and the patient comes to hold the same opinion. I can best explain my point by relating a case which we treated here in the hospital within the past few weeks.

One of our nurses in training is a widow with two children; both are away at a boarding school. One of them, aged nine, was sent home from school because of his intractable enuresis. The

mother presented him to us in the ward, and volunteered the history that he had always had this weakness, that he had been treated more or less for this condition since he was a baby. The teachers refused to keep him any longer, and she was advised to have his tonsils and adenoids removed; he had been previously circumcised without giving any relief. As the mother told the story she placed her hand on the little fellow's brow; they gazed affectionately at each other, tears were in both their eyes; they were both in great distress. The little fellow, too, saw the situation and, moved to emotion, said sadly that he had tried so hard to be cured; it was no use, he guessed he never would be cured, and then there was a duet of low sobs. At that moment I interfered. I fear I rudely separated them. I told the little mother in no uncertain tones what processes of thought were going on in my mind; I shamed and scolded the boy. The patient received a hypodermic of normal salt solution forthwith. He was given a red book, with instructions to mark down every accident that occurred, and he was promised that he would be cured in a short time. The mother was prevailed upon to remain away from the patient. He made an uneventful recovery. I could tell you of many similar cases.

The suggestive therapeutics may be varied to suit the occasion. The co-operation of the parents is essential. The plan of the patient's keeping a daily record, noting with good and bad marks each day's happenings, is helpful. The patient is also advised to void urine frequently during the waking hours, so as to exercise or strengthen the sphincter muscle of the bladder. The use of the faradic current frequently gives excellent results. One pole, the anode, is placed on the perineum, the other over the fundus of the bladder. Occasionally the patient is cured by one treatment, though, as a rule, the current must be applied several times a week over an extended period. I am convinced that when this treatment succeeds its effect is to be interpreted as a suggestive therapeutic remedy. Bimanual massage has been employed, one finger being introduced into the rectum, the palm of the other hand over the symphysis, and the bladder massaged in this manner.

The injection of 10 or 20 c.c. of normal salt solution into the epidermal sac, which is repeated two or three times a week and continued for four weeks, is said to be successful in 20 per cent. of cases. I cannot estimate the value of this treatment, as I have had no experience with it.

In place of these indirect methods of suggestion, hypnosis has been recommended and employed successfully in some cases. In the matter of drug treatment, belladonna is the favorite. The tincture of belladonna may be given to an infant one year old in 1- or 2-drop doses every three hours— $\frac{1}{2}$ grain of atropin dissolved in 1 ounce of water makes a solution of which a drop is approximately $\frac{1}{1000}$ grain; for a baby one or two years of age 1 or 2 drops may be given every three hours.

I am personally very skeptical about the drug treatment. While the enuresis may be temporarily helped by the use of atropin or belladonna, the symptoms produced by the drug may be alarming if pushed to the physiologic limit.

ANOREXIA IN INFANTS

THIS baby that I present this morning is thirteen months old; it had a normal birth, was nursed at the breast for a few weeks, and then placed upon a succession of infant foods. It did not do well and the foods were frequently changed. It is the first baby in this family. The mother is a very nervous woman, emotional, impetuous, unstable. The grandmother (the mother's mother) is everything that the mother is, only more so. She is emotional, imaginative, suspicious, distrustful, unstable. I do not know the paternal side of the family.

The baby did not gain in weight on the various food mixtures upon which it was fed, so the family broke up housekeeping about three months ago and started to travel over the country looking for health at the various health resorts. They took the baby to a sanitarium, where it was placed upon sanitarium treat-

ment—baths, hydrotherapy, dietetics, and the various methods of treatment that are employed at such institutions. Then the baby was taken to the seacoast, and there was placed under the care of a physician, who ordered fresh air, sunlight, and diet. It was given a light dose of citrate of magnesium daily. Finally, it lost so much weight that they concluded to retrace their steps homeward. They came home about a week ago and consulted me. The most prominent symptom which the child presents is anorexia—the baby won't eat; he not only has no appetite, but he has a repugnance to food; he pushes his bottle away, and then if he does take the food he frequently vomits.

Now in this particular case these people were in a great quandary; they consulted medical men and received medical advice, and were told that if the baby did not eat they should not force it. One doctor said, "How would you like to be forced to eat?" The mother very promptly said that she would not like it. The doctor said that it was the same way with the baby. The baby continued to lose in weight.

If we now examine the history record we find that the baby practically has no temperature (99° to 99.4° F.). The urine is acid, contains no albumin, shows an atypical reaction with Haines' solution, but one would say it was not sugar. The fermentation test, the use of the polariscope, have shown absence of sugar. The urine also contains acetone, but in starving babies you will find acetone which tends to disappear when food is taken. There are no casts, no leukocytes. The blood shows hemoglobin 72 per cent., leukocytes 14,600.

I will say, in passing, that this is a private case—the mother, of course, is not in the hospital, because we thought it best for the baby not to have her here. It is strange, too, that these cases usually occur in the so-called better families.

Looking at this child, you notice, first of all, a pale baby. It has rather a large head, the frontal portion is prominent and somewhat asymmetric, being somewhat larger on the left side; the fontanel is still open, sutures are closed for the most part. The baby is not able to sit up, as you notice; falls if he is not supported. His eyes appear large in comparison to his thin, emaci-

ated face. The mucous membranes are pale. There is nothing abnormal about the eyes. There are no enlarged lymph-nodes in the neck. The thorax is well formed and symmetric. One can very distinctly palpate a moderate rachitic rosary at the junction of the bony with the cartilaginous ribs. There is no thorax deformity, no marked depressions. The percussion of the thorax is normal, both anteriorly and posteriorly. As the child sits up you can see the rosary quite distinctly. The flesh is soft and flabby; the skin is elastic everywhere. Heart tones are normal, as is the respiratory note. The abdomen shows normal tonicity of the abdominal muscles, though it is slightly distended. The liver is easily palpable; the spleen cannot be felt. The tendon reflexes are present; there is no clonus, no Kernig, and no Babinski. He has erupted four teeth; the gums are rather swollen and soft; no discoloration.

What is the diagnosis in this case? What is the sickness from which this patient suffers?

VISITOR: Malnutrition.

ANOTHER VISITOR: Rickets; there is a degree of rickets.

DR. ABT: Yes, there is considerable malnutrition and a slight degree of rickets; neither, however, could be considered a satisfactory diagnosis.

VISITOR: Spasm of the pylorus.

DR. ABT: I think we can exclude spasm of the pylorus.

ANOTHER VISITOR: Chronic indigestion.

DR. ABT: There is something to that.

ANOTHER VISITOR: This baby has not had a chance; it's malnutrition.

DR. ABT: But this baby has one thing that stands out more prominently than anything else. Let us see how the baby eats, and then possibly you may change your opinion about the diagnosis.

VISITOR: How old is this baby?

DR. ABT: Thirteen months old.

VISITOR: The fontanel should be closed at the end of one year.

ANOTHER VISITOR: What is the character of the stool?

DR. ABT: As a matter of fact, the stools are very much improved at present. They are yellow, smooth, and without offensive odor. Here is some delicious oatmeal gruel with a little milk poured over it; we are trying to give the baby some, but he resents being fed. (Baby cries.) His every act is resistance against feeding; now he closes his lips and it requires considerable forcing to feed him.

DR. ABT (to Nurse): How did he take his breakfast, Miss S.?

NURSE: Fairly well.

DR. ABT: How did he take his supper?

NURSE: Not at all.

DR. ABT: I want you all to observe this, because this is what one actually sees in this group of babies. Normally he should not cry when he is to be fed, but this little fellow is sad when he has to eat. See him close his lips. If he makes a great struggle and gets worn out, he vomits after it. Try him with the bottle and see what an exhibition he will make. (Bottle given.) He closes his lips; he holds the bottle with his hands and pushes it away. He cries, refuses it, and struggles against it. What is the diagnosis after seeing this performance?

VISITOR: It is a spoiled baby.

DR. ABT: In a sense it is a spoiled baby, though one whose nervous system has been shattered to such an extent that we must consider the case pathologic. Many babies are spoiled, though only a few of them suffer from complete loss of appetite. Let us rather consider *anorexia nervosa*, also called *anorexia centralis*. As I have said before, these conditions in babies or young children occur under a variety of circumstances. A baby with a cleft palate sometimes suffers from *anorexia*. One of the worst cases I have seen was in a baby of fifteen or sixteen months, born with a cleft palate; the parents were healthy and in comfortable circumstances; the father was twenty or more years the senior of the mother. The child showed a retarded mental development and was moderately hydrocephalic. About the seventh month the baby refused food, and this condition of resistance against feeding and repugnance to food continued so that every feeding was a struggle and an effort. Every nurse who

attended the baby was worn out in a short time and simply could not remain on the case. It required superhuman strength to attend to that child. There was nagging on the part of the mother and father; they watched every bottle to see how much the baby took, and the mother herself could not feed the baby at all. It required absolute force.

In this case we thought that possibly there was a combination of causes that produced the anorexia. Possibly mental deficiency with hydrocephalus plus cleft palate. At any rate, when the baby was about fifteen or sixteen months old we considered the advisability of closing the palate. Things had come to such a pass that we felt if the palate could be closed we could force the feeding more effectually. This way it was like pouring water through a sieve—a great deal of the food would run through the nose. So we counseled a cleft-palate operation. The baby was duly anesthetized and operated upon, and for some reason (difficult to explain in these cases), whether from weak constitution or status lymphaticus, during the night it developed cardiac weakness, pulmonary edema, and the light went out.

We know anorexia occurs in acute diseases. For instance, a baby that falls ill with some febrile disorder loses his appetite. A baby that is attacked with pneumonia has great difficulty in taking food, and refuses it. The same is true of a baby who is ill with measles: he absolutely refuses food, especially when the eruption is at its height. But we may go further back than this. Infants not infrequently during the first days or weeks of life are very difficult to feed at the breast. This applies particularly to prematurely born or weakly infants who have insufficient strength to draw milk from the breasts. There is another group of babies who apparently are born without the sucking instinct developed; they neither eat to live nor live to eat. They constitute the class which Budin described as "lazy nursers"; the baby may take the breast for a few moments and then reject it, refusing entirely to return to the breast during the attempted nursing. In some instances the inability to nurse depends upon insufficient or entirely deficient breast milk—the so-called hypogalactia and agalactia; or, a baby acquires a repugnance to the breast because

the flow of milk is so slow that he becomes impatient and refuses it.

In cases where the processes of digestion are abnormal the appetite is likely to be subnormal or lost. Sometimes the infant's digestive function has been damaged by improper feeding or excessive feeding during the earliest period of life. Such infants acquire an atonic condition of the stomach with motor insufficiency of this organ. If the stomach in such cases be emptied four or five hours after the ingestion of food, it is found that only a small portion of the food has been emptied into the intestines. Not infrequently food is vomited mixed with thick, gelatinous mucus. Diarrhea alternates with constipation.

The most prominent symptom is the obstinate anorexia. The little patients have a positive repugnance to food. These cases are to be diagnosed by the history, the presence of gastric atony, and the evidence of gastro-intestinal disease.

Heubner, several years ago, described another group of cases under the name of "digestive insufficiency." This condition occurs in babies from the first to the third year, though it may persist into later childhood. The onset may be gradual, or it may occur suddenly, with fever and general symptoms of malaise, fretfulness, constipation, loss of weight, and anorexia. The appetite continues to diminish; it is lost during the exacerbations. The course is characterized by a recurrence of attacks of indigestion.

The patient is, as a rule, underweight, feeble, and pale. He has been progressing normally for a time, though the appetite has been poor. Suddenly, out of a clear sky, without any change in diet, a so-called "*catastrophe*" occurs. The abdomen becomes tympanitic, diarrhea occurs with the evacuation characterized by a pungent, sour odor, considerable gas, with undigested food particles. The patient loses weight, becomes more pallid; after several days or weeks the patient rallies, the appetite returns to some extent, and improvement is taking place, when another *catastrophe* occurs. It is not clear whether these cases depend upon some congenital weakness of the organism and of the digestive function, or whether they are caused by repeated insults to

the digestive function resulting in a weakness of the process and recurrent attacks of digestive insufficiency.

I have seen a number of cases which correspond to Heubner's description of digestive insufficiency. It seems that the digestive processes remain normal for a time and then a condition of fatigue and digestive exhaustion occurs, resulting in an attack characterized by the collapse of the normal physiologic functions of digestion and metabolism.

In certain nutritional disturbances the appetite suffers. In some cases of rickets anorexia may be a prominent symptom. An infant suffering from scurvy refuses food, as a rule, though he frequently takes orange juice with avidity.

We must not forget other local causes for anorexia. I again call your attention to cleft palate and hare-lip, retropharyngeal abscess, occlusion of the nasal passage, stomatitis, as mechanical obstacles to feeding. Congenital double-sided facial paralysis may offer a serious obstacle to nursing or sucking. Tuberculosis and, more particularly, meningitis offer obvious causes for disturbed appetite or complete anorexia. Idiocy and cretinism may contribute to a more or less complete loss of appetite. We must assume that in some cases there is an organic brain lesion which prevents the occurrence of the physiologic sucking reflex. This may occur in injuries to the brain during the process of birth.

Babies show peculiarities of taste. A young baby that refuses water will take it if it is sweetened. Another young infant objects to sugar in the food, but takes its milk eagerly if less sweetened. You have already heard from mothers in the clinic that the babies and young children refused cereals, but would eat cracked wheat, shredded wheat, puffed rice, or grape-nuts. The same is true of vegetables. The mother or nurse reports that the child prefers this or that vegetable, but refuses most of them. I think, in most instances, this is a matter of training. Most children can be taught to eat what is good for them if the discipline is anywhere near correct.

With reference to the case that we have examined this morning, I feel justified at least in making a provisional diagnosis of anorexia nervosa.

This condition was first described by Lasegue in 1873. In the following year Sir William Gull, writing independently, described the same condition under the name "anorexia nervosa." In 1894 Soltmann described the first cases in children, referring to them as anorexia cerebialis or corticalis. Forchheimer described several cases in children in 1907. One of his cases, a boy thirteen years of age, died of starvation. J. P. C. Griffith also described several cases the same year. The case reports are not numerous, though I am certain they are not of uncommon occurrence.

Remember the history of the case; the parents are well situated in life, neurotic, self-centered, inclined to be intellectual, though lacking somewhat in mental equilibrium. These babies, then, come from nervous families. Not only has this baby inherited a nervous stigma or strain, but the environment remains tense. When the mother first takes the baby on her arm every tremor and all the agitation of soul and body are transmitted to this baby. She can't help it, and these tremors are there constantly when she holds her baby. The baby is in a tense, nervous atmosphere; it never has a chance to relax. Twenty-four hours after the birth of the baby the neighbors came trooping in as they would to behold a soldier lying in state, with great noise and bustle, and this continued more or less. Every new visitor who arrives is shown the baby; every time the baby has a new dimple the town folk are called in to see it; every time the baby erupts a new tooth all the fond friends come over. Everyone wants the privilege of holding the baby, kissing the baby, and, not only that, the mother sits down with it, sometimes she picks it up, sometimes she talks to it when it is asleep, sometimes she starts the Victrola in action so that he may become a music lover and develop an artistic temperament in later life. It is true—these things are all done. Sometimes she sings to it or she recites a classic so that it may develop a literary taste. All of this occurs in the environment in which this baby lives. The father is no better; men are influenced also by the environment in which they live. A calm, placid man, who ordinarily did not know much about babies, who was willing to let them live

and grow up, becomes influenced by his wife. He wants to please and be attentive, so he too picks up the baby; throws the baby half-way to the ceiling, tickles it under the chin, tries to make it laugh and have a good time, and all this is at the expense of the nervous system of the infant; and then by and by baby becomes worn out. Every human being after a time will suffer fatigue, and if sufficient rest is not guaranteed to this human being, if the fatigue continues too long a time, then pathologic conditions result. This baby is already neurotic, and its environment conduces all the more to neurasthenia.

This baby begins in a short time to show repugnance to food; he refuses his bottle and pushes it aside; he refuses solid food; if the feeding is persisted in, he vomits; he makes a great struggle. You saw the baby here. He is accustomed now to resist, and up to this point his resistance has been successful. He has fought the nurses and parents until now he eats when he pleases or not at all. He has been treated with various kinds of medicines without any result. As I said a moment ago, these babies refuse to feed, or if forced, they vomit. They become pale, they lose in weight, the bowels are likely to be constipated; muscles become relaxed; sitting, walking, standing are much delayed. The normal development of the body is much retarded. For instance, in this baby there is a failure of the fontanel to close. In general, it suffers from a marked malnutrition and a slight degree of rickets, which I consider of secondary importance. Most of these cases depend upon a functional neurosis, usually inherited. Occasionally, of course, there are some severe nervous conditions at the base of the trouble. Occasionally meningitis, or cerebral tumor, or some severe pathologic process is at the bottom of it, and for that reason I advise you not to pass too lightly over these cases; not to jump at conclusions. I would advise you to examine every case carefully and to decide every case on its merits. I would advise you, then, to make careful examination of the nervous system; of the motor reflexes of the body; of the general physical condition of the baby before arriving at the diagnosis.

The prognosis is not altogether bad so far as life is concerned in cases of anorexia nervosa, though some of these cases die

directly from an intercurrent disease and some from starvation. Whether these babies once being neurotic or hysteric always remain so is somewhat difficult to say. It seems to me I have seen some of them make complete recoveries. Some of them seem to acquire stable nerves.

The treatment of these cases is important. What shall we do with these babies? In the first place, something depends upon the age of the baby. If it has already erupted teeth, we must first pay attention to the interval at which to feed it. Shall we feed them frequently? I answer, Feed them at long intervals. Why? Because each feeding is such an effort that the process not only wears out the baby but the nurse, and it would require three nurses, if you were to feed these babies at short intervals. Another thing, let the child become slightly hungry for food. Feed the baby at long intervals.

Now, what shall we feed the baby? If the child is eight or ten months old, diminish the quantity of milk somewhat or diminish it materially and substitute other food. These babies, as a rule, do not desire milk. And then, too, it is better to give them food that is of smaller bulk. Better to give food that is more concentrated. So that if these babies receive 20 to 26 ounces of whole milk a day, and then other food be added to make up the deficiency, I think the best results will be obtained.

These babies may have cereals, well-boiled cereals, or they may have strained vegetables, especially at the eighth or ninth month. It is advisable to give them orange-juice, because in some cases the baby has been deprived of food for so long that there may be some nutritional disturbance allied to scurvy. So it is a good plan to give them orange-juice and plenty of it. Then broths are sometimes tasty—sharpen the appetite a little bit—and if they are combined with vegetables and cereals they have some nutritive value.

These babies may be fed at 6 and 10 A. M.; 2, 6, and 10 P. M. Not infrequently it is a splendid scheme to give them only three feedings a day—8, 12, and 6 o'clock. Let them wait a long time for their breakfast. If the feeding is extremely difficult, both to the baby and nurse, feed it three times a day.

Having decided on the diet and the intervals of feeding, the next thing is to secure the co-operation of somebody that will be the "man behind the gun." The success depends upon somebody who will faithfully carry out orders. The mother can't do it, because if the mother could do it she would have done it long ago. As a rule, the mother's presence seems to irritate these babies. I have seen babies who would not eat when the mother was in the room. You require a nurse to feed such children. She must be absolutely reliable, and then she must have perseverance; she must persist until she succeeds. She must be quiet.

In the case I showed you this morning, when I made my first examination the mother and grandmother of the baby were just shouting at the child and shouting at each other. Everybody made everybody else nervous. You could feel the tension in the air. These babies need a quiet environment; they need very little talking to; an occasional word of command from the nurse, and then the rest is action. I tell these nurses, "Don't talk to the baby; don't sing; don't stimulate him; just show that you mean business." You saw how difficult it was to feed this baby. We sometimes use a Breck feeder. I will show you what I mean by a Breck feeder (Fig. 91). Sometimes we feed the baby with a stomach-tube, or in other cases we feed it with a spoon.

This point I want to emphasize: With babies that have been sung to, for whom the band has been played and music has been invoked, stop all that; put the baby in a quiet environment, show him that feeding is strictly a matter of business, and the chances are that if you persist for a little while the baby will be cured. Sometimes we give these babies a little medicine; minute doses of arsenic two or three times a day; that is, 1- or 2-drop doses of Fowler's solution, or 1 or 2 drops of dilute hydrochloric acid three times daily after feeding, both well diluted with water.

The most important thing, however, is to get these babies out of their environment. No matter how much you may try, it is difficult to cure such babies in their homes. That tense atmosphere of which I spoke is almost impossible to control. You can't re-educate the parents in a short time; you can't re-educate the grandparents, who are in constant attendance upon the baby.

You can educate or re-educate the baby, and I am very confident with this baby here, removed from the parents, the work is possible.

I put a faithful, reliable nurse on this case. The first night after she had been there she came to me crying, and said it was impossible, that she could not feed the baby; she could not force the baby, for if she did the baby's mother and grandmother flew into a terrible passion. So you see the difficulty. The most important thing is to take the baby away from its environment.



Fig. 91.—Breck's feeder.

Remove it somewhere or isolate it in the home, and then begin this matter of re-education, forcible feeding, rest, and, as nutrition improves, as the general condition of the baby improves, as the health and strength increase, the baby's appetite will return.

We have a great many of these cases; many have come from afar; many that have been handled in every possible way, and we have proved for ourselves that the treatment is:

A splendid, quiet nurse, one who understands the work, and placing the baby in quiet surroundings with a simple process of feeding. It is a very interesting group of cases.

CLINIC OF DR. ROBERT B. PREBLE

ST. LUKE'S HOSPITAL (AT BEDSIDE)

THE RHEUMATIC TRIAD. A DOUBLE MITRAL LESION IN A BOY OF SEVENTEEN WITHOUT SUBJECTIVE SYMPTOMS

THIS boy is seventeen years old, and came into the hospital a couple of weeks ago complaining of headaches, sore throat, and fever. He had not been feeling well for about a month, and on Monday afternoon began to feel worse, with chills and headache; the muscles all over his body ached, especially in the back and arms. On Tuesday night he vomited and his throat began to hurt him; on Wednesday he had nosebleed, and for the past few days has been having pain in the joints.

So far as the previous history is concerned, he had a severe sore throat when twelve years of age. In March, 1914, he had rheumatic pains in the knees without any swelling; the pain involved other joints, but not so severely. In April, 1914, he was told that he had some trouble with his heart.

When he came in the temperature was 102.2° F.; what would you think about such a case as that?

VISITOR: I would think of the attack of tonsillitis furnishing the infection.

DR. PREBLE: Yes, the attack of tonsillitis probably furnished the infection. The temperature ran 102.8° F. the day after admission; the next day it was 101.6° F.; the next day, 101° F.; the sixth day it was 99.6° F., and since then he has been practically afebrile, although occasionally he has had a temperature of 99.2° or 99.6° F. What other thing would occur to you aside from tonsillitis or arthritis?

VISITOR: From the early part of the history one might think of a beginning typhoid—the nosebleed.

DR. PREBLE: Yes, but nosebleed occurs with other things; what would you think about it, Dr. Dinneen?

VISITOR: I would think of an acute endocarditis.

DR. PREBLE: Yes, of an acute exacerbation of an old endocarditis which his doctor recognized. Does that sound reasonable?

VISITOR: Yes, sir.

DR. PREBLE: It happens not so very uncommonly; I showed you a little girl with a decided heart lesion who knew nothing at all about it. (To Patient): What work do you do?

PATIENT: Office work.

DR. PREBLE: Do you take any exercise?

PATIENT: Yes sir; at the Y. M. C. A.

DR. PREBLE: What kind of exercise do you take there?

PATIENT: All kinds of athletics—running and basket-ball.

DR. PREBLE: You don't get out of breath any sooner than the other boys?

PATIENT: No, not at all.

DR. PREBLE: Did you know that you had anything wrong with your heart at that time?

PATIENT: Yes, but I had no symptoms.

DR. PREBLE: When he came in he had a red, sore-looking throat, and a culture revealed some diphtheroid bacilli, but that quickly quieted down, and the pains in the back and arms and legs lasted only a short time. (To Patient): And now you have been out of bed for how long?

PATIENT: About four days.

DR. PREBLE: When he came in the blood-cultures were negative. (To Doctor): What do you see, Doctor?

VISITOR: The apex-beat lies here about the fifth interspace and is rapid.

DR. PREBLE: And how far out?

VISITOR: Within the nipple line; there is rather a diffuse area of pulsation all over the precordium; there is no retraction and no inequality in the size of the chest.

DR. PREBLE: And the heart is beating regularly.

VISITOR: It is a little rapid.

DR. PREBLE: In general, it runs along about 80, smoothly and regularly. Can you see the closure of the second pulmonic?

VISITOR: Yes; I can see it right there (indicating).

DR. PREBLE: Can you see the valves close?

VISITOR: I would have to count, but it is easy to feel it, I think.

DR. PREBLE: Can you feel the closure of the second pulmonic?

VISITOR: Yes, very easily.

DR. PREBLE: Easily, but you are not quite sure that you can see it close?

VISITOR: No, I am not sure of that, but I can feel it all right.

DR. PREBLE: Very easily; I think you can see it, too; it is very easily palpable. Now what can you feel over the apex-beat? Is such a palpable closure as that pathologic?

VISITOR: Yes, I think it is, but in such a young person it might not be.

DR. PREBLE: He is well beyond the age of puberty and his second pulmonary ought not to show the infantile accentuation. (To Doctor): What can you feel over the apex?

VISITOR: It is circumscribed and there is a thrill.

DR. PREBLE: Is it systolic or diastolic, or both?

VISITOR: It seems to be diastolic, just before the systole.

DR. PREBLE: A presystolic thrill; what is the second thing that you feel, a tone or a thrill?

VISITOR: I don't know; there is a thrill there, but I am not sure which is which. They give impulses to the hand which are a good deal alike.

DR. PREBLE: See if you can make that out, Doctor S.

DR. S.: I can feel the mitral valve.

DR. PREBLE: Can you feel the first tone?

DR. S.: Yes.

DR. PREBLE: I think I can feel a little bit more than that; the easiest thing to pick out is the first tone, and there is a pre-systolic and systolic thrill. (To Patient): Please sit up. (To Doctor): Try it in that position and see if you can get more.

DR. S.: That brings it out better; instead of a tone you get kind of a rough feeling like the heart slipping. It is not a well-defined thrill.

DR. PREBLE: It makes them both more pronounced when he is sitting up; you can get a tone and then a thrill. Can you feel the tone at all, Doctor?

DR. S.: No; I don't think I can.

DR. PREBLE (to Patient): Get up and walk quickly down the ward and back. (Patient obeys.)

DR. PREBLE: Now see if you can feel it.

DR. S.: It is much easier to feel now.

DR. PREBLE: It very often happens that these are difficult to determine when the heart is quiet, and they become more pronounced when the heart is stirred up a little. So we will say that over the apex one can feel a systolic tone preceded and followed by a thrill, and that the second pulmonary tone is very easily palpable. Now let's percuss that out and see what we find.

VISITOR: It seems to be pretty low; I would say that the heart is enlarged both ways.

DR. PREBLE: That is, transversely and to the left and upward. It's pretty high up there; that's the lower border of the second rib, isn't it? That's the liver dulness at the lower border of the fifth interspace close to the sternum. The apex-beat is right here (indicating); the cardiac dulness which starts at the border of the liver, $\frac{3}{4}$ inch to the right of the sternum, crosses the fourth rib at the junction of the rib and sternum, then crosses the right border of the sternum at the upper border of the third rib and down and out to the apex-beat, which is behind the fifth rib at just about the midclavicular line. Have we got enough to make a diagnosis?

DOCTOR: It is claimed that a heart lesion can be diagnosed upon inspection and palpation; any heart that is enlarged transversely would seem to be a double lesion.

DR. PREBLE: What do you mean by a double lesion?

VISITOR: A double mitral.

DR. PREBLE: Not necessarily; a single mitral will do it too. What data have we now?

VISITOR: We have the history of an infection.

DR. PREBLE: Yes; he has had attacks of sore throat since he was twelve, some with rheumatism and some not. At no time has he had any symptoms of decompensation, even in so strenuous a game as basket-ball.

VISITOR: It has been a slowly progressing condition.

DR. PREBLE: We don't know about that; we only know that the earliest opportunity for endocarditis, as nearly as we can get it from the history, was when he was twelve; that he has had no cardiac symptoms to his knowledge. We know that a cardiac lesion was diagnosed a year ago. On inspection, what do we see?

VISITOR: The apex-beat displaced toward the left.

DR. PREBLE: Yes, and we think we can see the closure of the pulmonic valve.

VISITOR: On palpation we get a systolic thrill.

DR. PREBLE: We get a presystolic and a systolic thrill and an accentuation of the pulmonary second tone. Then, on percussion?

VISITOR: On percussion we find a heart which is enlarged to the right.

DR. PREBLE: A heart which is transversely enlarged, mostly to the right, and upward to the upper border of the third rib. Have we got data enough there to make a diagnosis?

VISITOR: I think so.

DR. PREBLE: We have data enough to say that he has a heart lesion; how much further can we go?

VISITOR: On just the physical findings I think one can diagnose part of the heart lesion at least, but with a double mitral one would expect more enlargement to the left.

DR. PREBLE: Not necessarily.

VISITOR: I would say that he had a double mitral.

DR. PREBLE: What are the things that speak for a mitral?

VISITOR: The enlargement of the heart transversely, particularly to the right.

DR. PREBLE: The enlargement of the heart to the right, the accentuation of the second pulmonic and of the first tone, the enlargement upward, and what other things?

VISITOR: The thrills.

DR. PREBLE: The systolic and presystolic thrills. All of those things speak for a mitral lesion; insufficiency or stenosis, or both?

VISITOR: Both, I should say.

DR. PREBLE: Certainly; with the presence of the palpable first tone and with the diastolic thrill we have to say that it is not pure insufficiency; there is also a stenosis. It is not necessary at all to use your stethoscope. It is perfectly certain that the boy has a heart lesion and that it is a double mitral. Listen to it now and see how it sounds. Would you say it was compensated or uncompensated?

VISITOR: It has been compensated up to this time.

DR. PREBLE: Yes; any boy who can play basket-ball in spite of a double mitral certainly has no decompensation. What do you think of the wisdom of his doing it?

VISITOR: I should think he ought not to do it.

DR. PREBLE: No, it appears to me to be a very unwise thing, in spite of the fact that he has had no trouble from it. What exercises would you allow him?

VISITOR: Moderate exercise, such as walking, tennis, etc.

DR. PREBLE: In a general way, it is not wise to allow a boy to play basket-ball or tennis, or any game of that sort, because the competition is apt to lead to overstrain; but almost any exercise which he can take without that element of competition is all right. Working with pulleys, weights, Indian clubs, horseback riding—any of those sports are all right, and he should be encouraged to do them within reasonable limits. But practically none of the ball games can be entered into without risk. What would you say about medicine ball?

VISITOR: I don't think it would be very good for him.

DR. PREBLE: He might play with a small medicine ball, but not a very large one, that is too hard work. (To Doctor who has been examining with stethoscope): What do you hear?

VISITOR: At the apex I hear a murmur, and it is transmitted into the axilla.

DR. PREBLE: You can hear the systolic and diastolic murmur; can you hear anything else?

VISITOR: The second pulmonic is very loud.

DR. PREBLE: The second tone is accentuated. Can you hear anything else?

VISITOR: I could hear the first tone.

DR. PREBLE: What do you hear at the base?

VISITOR: The second pulmonic was loud and snapping.

DR. PREBLE: Can you hear murmurs at the base? You see, a boy like this might easily have an aortic lesion.

VISITOR: I could hear the two tones at the aorta.

DR. PREBLE (to Patient): Please go up and down the ward again. The other day that first tone was so loud that one could hear it through his clothes, 2 inches away from his chest, but it is not nearly so loud as that today. Perhaps this exercise will stir it up a little. (Patient returns, and Dr. Preble listens to his heart.) That brings out the first tone and makes it very loud, so you can hear it through the air. So now we will make this sort of a diagnosis: He has a double mitral lesion, well compensated, and as a result of the recent infection there is an acute exacerbation. He has some bad teeth; what would you say about those?

VISITOR: His tonsils are not bothering him.

DR. PREBLE: Not now, but when he came in he had a temperature of 103° F.

VISITOR: After the acute disease has subsided he ought to have his tonsils taken out and his teeth fixed up.

DR. PREBLE: Yes, he should do both of those things. Does he need any medication so far as his heart is concerned?

VISITOR: It wouldn't do him any good.

DR. PREBLE: No; on the contrary, if you give him active medication it might do him harm. Would you give him a tonic?

VISITOR: The value of a tonic is doubtful.

DR. PREBLE: I don't remember his blood-picture, except that he had about 16,000 whites when he came in; his reds are a little shy. He had 30,000 whites on one occasion, according to the history. The urinalysis showed a little albumin and a few hyaline and granular casts. At that time he had an acute infec-

tion; the urine ought to be examined repeatedly before he is dismissed from the hospital, for, while the albumin and casts are probably only a part of the infection, we want to be sure of it. How about his work?

VISITOR: I should think the kind of work he is doing is all right.

DR. PREBLE (to Patient): How far did you go in school?

PATIENT: Through the eighth grade.

DR. PREBLE: What kind of work do you do in the office?

PATIENT: I keep some books and do a little bit of everything.

DR. PREBLE: I think it would be a wise thing for you to go to school some more. How long have you been at work?

PATIENT: About three years.

DR. PREBLE: It seems to me that if it is in any way feasible for you to go to school three or four years more it would be better for you. You could get a better job then, couldn't you?

PATIENT: I don't know; I might.

ACUTE CATARRHAL JAUNDICE

DR. PREBLE: What do you think of that boy?

VISITOR: He looks extremely jaundiced.

DR. PREBLE: I should call it a mild rather than a high grade of jaundice. Do you suppose it has been going on for a long time or a short time?

VISITOR: I can't tell.

DR. PREBLE: You can't tell with certainty, but you can say that if it has been going on for weeks or months then you must conclude that it is not the ordinary type of jaundice that you see in men of his age. (To Patient): How old are you?

PATIENT: Twenty-five.

DR. PREBLE: At that time in life there is only one kind of jaundice that is common, and either gives rise to very intense jaundice or jaundice of very short duration. What is that?

VISITOR: The acute catarrhal jaundice.

DR. PREBLE: Yes; that is either very intense or very brief in duration. In men of his age it is usually very transient or much more intense than this. Why is that? What happens in an acute catarrhal jaundice?

VISITOR: The common duct becomes plugged.

DR. PREBLE: Yes; the common duct becomes plugged and we have the resultant pigmentation. As a general thing, how extensive is the plugging?

VISITOR: It is usually complete.

DR. PREBLE: Yes; it is usually complete. It is difficult for us to imagine a case in which the common duct was not absolutely plugged with the patient becoming jaundiced very rapidly. While one can conceive of a case with imperfect plugging which permits slight leakage, that is not the rule. The obstruction is usually complete, and the jaundice is very intense unless the circulation of the bile is re-established. (To Patient): Tell us about this trouble.

PATIENT: At first I was working every day, and one day I started to eat dinner and nothing smelled good to me and I didn't feel like eating, but I thought it was the hot weather and kept on for a few days, and then a week ago Thursday the food began to come up on me. As soon as anything got in my stomach it would come up.

DR. PREBLE: How long did that last?

PATIENT: Until Sunday.

DR. PREBLE: Did you have any pain?

PATIENT: No.

DR. PREBLE: Did you have any fever or any chills?

PATIENT: No; a customer came in on Saturday and said, "You are all yellow," and that was the first I knew of it.

DR. PREBLE: At what time on Thursday did you notice that there was something wrong?

PATIENT: On Thursday morning. My bowels wouldn't move.

DR. PREBLE: How long a time from the time he began to feel sick until he was jaundiced? He became jaundiced some time on Saturday morning.

VISITOR: It might have been present before.

DR. PREBLE: It probably was, and somewhere along about forty-eight hours after he noticed something was wrong with him he discovered he was jaundiced. Is that rather a short time or a long time?

VISITOR: Rather short.

DR. PREBLE: The jaundice usually begins to appear from twenty-four to thirty-six or forty-eight hours when the plugging of the duct takes place suddenly. With a stone you know just when the obstruction occurred. He became jaundiced about forty-eight hours after the first symptom. (To Patient): Did you notice the stools on Saturday?

PATIENT: No; on Sunday.

DR. PREBLE: What color were they then?

PATIENT: They were dark.

DR. PREBLE: Have they been light at any time?

PATIENT: Not until today; they are a little lighter.

DR. PREBLE: What color are they now?

PATIENT: I don't know exactly—just about the color of light beer, I guess.

DR. PREBLE: But you have not had any pain?

PATIENT: No pain at all.

VISITOR: I thought they usually had pain.

DR. PREBLE: They are apt to have some stomachache, but it is not always present. Nausea and vomiting are not always associated with the disease either. He feels pretty good and doesn't understand why we keep him in bed and make him miserable.

PATIENT: With nothing to eat and just lots of water!

DR. PREBLE: The laboratory report on the stools is not finished yet. He has a nice looking, yellow body. Suppose you examine him and see what you can find.

VISITOR: There is jaundice all over, and there seem to be some red blotches on the side of his face.

DR. PREBLE: I suspect that is just an ordinary, low-grade acne and has nothing to do with his jaundice. He is uniformly jaundiced, not an extreme grade, but rather mild. The general nutrition is good, there are no evidences of emaciation, the muscles are firm.

VISITOR: The pulse is moderately slow, but regular, and the arteries are not particularly hard.

DR. PREBLE: The pulse has not been particularly slow; the chart shows 87, 88, 80, 72; there has not been any bradycardia.

VISITOR: I make it 66 now.

DR. PREBLE: It may have come down a little. There is no adenopathy at all.

VISITOR: The pupils react promptly to light; I don't notice anything else in particular.

DR. PREBLE: No; so far as inspection of the body goes, aside from the jaundice, he is in pretty good shape. His habits are no worse than the average. He has been in the hospital before, was here for six months with pneumonia and empyema. However, that is old history and has no bearing on this at all. He is just a healthy young fellow who has been through that experience. Just examine the abdominal region and see if you find anything. So far as inspection of the abdomen is concerned, it is perfectly negative; there is no distention.

VISITOR: There are apparently no tumors; I can't feel the spleen; the liver is palpable.

DR. PREBLE: No; his spleen can't be felt at all; one can feel the liver, but there is no demonstrable distention of the gall-bladder; the liver can be felt only on deep inspiration. Palpation of the abdomen elsewhere is normal. All that can be said is that on the abdomen, aside from the jaundice, there is nothing that can be felt or seen. The urine shows bile, but whether or not the stools contain bile I cannot say, for the laboratory reports are not ready. However, from his description of them one would say that they did. The blood shows a normal red and a normal white count, the hemoglobin is a little lower than you would expect it to be; the differential count is normal. What would you think about that?

VISITOR: The history and negative findings, except for the pigmentation, would make me think it was simply a catarrhal jaundice.

DR. PREBLE: Is the matter of any consequence except for the discomfort?

VISITOR: Yes; we don't know how far it will ascend the bile tracts, and it may get into the gall-bladder or liver and set up other trouble.

DR. PREBLE: Yes; let's be just as pessimistic about it as we can. It may not be an acute catarrhal jaundice at all; it may be the initial stage of an acute yellow atrophy. So far as he has been under observation he has shown no infection, and while that is possible, the changes are very slight; so slight that it is not necessary, so far as he is concerned, to dwell upon them. Catarrhal jaundice is a nuisance, but he is getting off rather easier than usual, at least so far as discomfort is concerned. Thursday he felt bad, Friday he felt bad, and Saturday he didn't feel very good, but since then he has felt all right. He didn't even suffer the usual discomfort that many of these cases endure. Even the skin irritation has been spared him; the urine is not bad. It seems to be a simple catarrhal jaundice with the man suffering less than usual. What are you going to do for him?

VISITOR: Keep him in bed now.

DR. PREBLE: Why?

VISITOR: You probably want to put him on a light diet.

DR. PREBLE: We put him not on a light diet, but on a starvation diet; he is getting nothing at all but water.

PATIENT: And chewing gum.

DR. PREBLE: Well, I don't mind the chewing gum. We put him on a starvation diet, and that is the great reason for keeping him in bed, for then he feels the lack of food less. What is the idea of keeping him on a starvation diet?

VISITOR: The process is supposed to be localized, but sometimes there is a dietary phase when a slight infection may appear.

DR. PREBLE: That is what we are after; to put his gastrointestinal tract at as complete rest as we can and, therefore, we take away all of its work; no food at all. How long will we keep that up?

VISITOR: Until he shows marked improvement.

DR. PREBLE: No, we can't do that, for sometimes these fellows stay sick for several weeks, and we can't starve them to

death. We usually keep them on this diet for five or six days or a week, and it is not likely that a longer rest than that will be necessary. If we wanted to we could keep him on a starvation diet for ten days or two weeks without hurting him any, but that is not really necessary. How about the water; is that keeping his canal at rest?

VISITOR: The water does not stimulate at all and it flushes.

DR. PREBLE: Yes, and we have to give him water; he can't go without that for very long, and even though it may be that the introduction of water has some effect in stimulating peristalsis, it can't be withdrawn for any length of time. Of course, it could be given per rectum, but that is a nuisance and altogether unsatisfactory, and it is much easier to let him have it by mouth. What temperature should the water be?

VISITOR: I suppose if it could be given at a temperature of about 90° F. it would not irritate at all.

DR. PREBLE: Why do you specify 90° F.?

VISITOR: Because that is nearer the temperature of the part; that is, probably about 98° F.

DR. PREBLE: It is probably about 105° F. deep in there, and it is better to give him the water about 103° or 105° F.; better than either too cold or too hot.

VISITOR: What do you put in the water to make it palatable?

DR. PREBLE: Nothing but H₂O. Water really isn't unpalatable when you get used to it. Their tendency, of course, is to drink cold water, but it is best to give it to them warm and it is best to give a specified amount. He gets 8 ounces every hour and as much more as the nurses remember to give him; no food at all. How about medicine?

VISITOR: Calomel is used to some extent.

DR. PREBLE: Yes; we usually give these patients a little dose of calomel followed by a saline. After that there are a variety of things that are quite customary. How valuable they are, I think, is a question—sodium sulphate, urotropin, and other things. How long is that jaundice going to last?

VISITOR: It will probably last six or seven weeks. It takes a long time for the pigment to leave the skin.

DR. PREBLE: How long it will be until the obstruction of the duct is relieved one can't tell. It may be a few hours, or a few days, or a few weeks. If the obstruction is overcome within the first few days the jaundice disappears quite rapidly, for the discoloration is due to the blood-serum. If the pigment is once deposited it usually takes about three weeks for the yellow color to disappear, although it can be hastened by the use of hot baths, rubs with a Turkish towel, and so on, to stimulate the exfoliation of the skin.

VISITOR: We often see such cases in people who are up and around all the time, and in those instances what should be done?

DR. PREBLE: In individuals of this age any other kind of a jaundice than a catarrhal jaundice is quite exceptional. Of course, one may have a cirrhosis of the liver at any time, and many heart cases in which the strength is below normal are subicteric, but at least 9 out of 10, or 19 out of 20, or maybe 49 out of 50 are catarrhal, and these are handled in just the same way. If the jaundice is of some other type, if it is due to failing compensation, that is another thing, and if it is due to cirrhosis, that is still another, but for the great bulk of them at this period of life this is the way they should be taken care of—give them a chance to rest up and starve them for a little while and give them lots of water.

CLINIC OF DR. WALTER W. HAMBURGER

COOK COUNTY HOSPITAL

ABDOMINAL PAIN. DIFFERENTIAL DIAGNOSIS. THE CAUSATION OF THE "HUNGER-PAIN." THE "PYLORIC SYNDROME" OCCURRING IN A CASE OF TERTIARY SYPHILIS. SUCCESSFUL TREATMENT BASED ON ACCURATE DIAGNOSIS. SOME DIETARY PRINCIPLES INVOLVED IN THE MANAGEMENT OF DISEASES OF THE STOMACH, WITH PARTICULAR REFERENCE TO THE INFLUENCE OF WATER ON GASTRIC SECRETION AND MOTILITY.

STARTING with the case this morning we shall attempt to present patients illustrating disease conditions with various types of abdominal complaints, such as abdominal pain, vomiting, nausea, indigestion, dyspepsia, etc., and shall attempt to analyze the cases from the standpoint of this principal subjective complaint. This method I believe is of value, particularly from the standpoint of differential diagnosis, and ultimately, of course, from the standpoint of medical management and treatment, because the successful treatment of these cases depends largely on the accuracy of the clinical diagnosis.

This patient is a Russian, a polisher by occupation, twenty-eight years of age. He entered the hospital on the 12th of July with the principal complaint of abdominal pain. The pain was centered around the umbilicus and in the epigastrium, and comes on about fifteen minutes after eating. The pain is of a burning, sore character, and an hour or so after eating it becomes more severe. It is not referred to the shoulder nor down to the pelvis, but at times runs around to his back. The patient has a sensation of fulness after each meal which sometimes takes the place of the pain. He sometimes induces vomiting by putting his

finger down his pharynx to relieve him; he does not vomit otherwise. He has never vomited blood. The pain is not relieved by milk; he has never used soda. In addition to the pain he complains of anorexia, but this is rather from fear of increasing his pain than from true loss of appetite. He is afraid to eat because of the after-effects. He is moderately constipated; has lost about 40 pounds in weight in the last six years. He complains of tenderness upon pressure on either side of the umbilicus, which is distinctly sore and tender. His stools are thin and loose. The patient speaks very little English, and this history was obtained through an interpreter. This illustrates the difficulty of obtaining a really accurate gastric history from patients of foreign birth because of their inability to understand the different phases of the things you are asking about. One day he will tell one thing and the next day will negate this entirely, but he sticks to his story of the pain.

The onset has been over a period of six or eight years; at that time he noticed that he was losing his appetite and was having pain as described above. His appetite became further impaired, partly because of anorexia and partly because of fear of causing increased pain. He returned to Europe shortly after the onset of the trouble and served as a soldier, where he was fed coarse, dry bread and very little meat. He later returned to the United States and his condition became worse again, although it had improved somewhat while he was in Europe. Since the onset of his trouble he has lost about 40 pounds.

To sum up, he complains of pain coming on fifteen minutes to an hour after eating, increasing in intensity, and relieved to some extent by food. This has been developing for six or seven years, and the pain has been so severe as to cause him to fear to eat. Returning to Europe and living on very little meat and coarse, dry bread the pain was less severe, but on his return to America he again becomes worse. We might outline it in this way:

- (1) Pain fifteen minutes to one hour after meals.
- (2) Loss of weight—40 pounds in about six years.
- (3) So-called anorexia; a combination of loss of appetite and fear of pain from eating.

The complaint we are particularly interested in this morning is his abdominal pain. He has never had nausea or chills; has not had radiating pain; the pain is not cramp-like or colicky. It is a burning pain, comes on fifteen minutes to an hour after eating; increases in severity. His complaints have not been progressive, but are stationary, and the pain is relieved somewhat by food, but is not relieved by milk, and relief from soda is questionable.

INTERN: In regard to the question of relief by food; is it not the food that brings it on?

DR. HAMBURGER: Yes. The pain comes on about fifteen minutes after eating, as a rule, but we are not sure whether the next meal intensifies it or not. Has it continued until the next meal or has it disappeared spontaneously? We want to find out whether food relieves or intensifies it.

INTERN: Is the relief by vomiting important?

DR. HAMBURGER: He has not vomited except by putting his finger in his pharynx; he has no spontaneous vomiting, but when he induces vomiting, that gives relief.

INTERN: Was there blood in the stool?

DR. HAMBURGER: He says that his stool was black, but we cannot conclude from that alone that it was blood. It might have been the effect of medicine.

(Continuation of history.) *Previous Diseases*.—He was sick for six weeks with chills and fever about seven years ago, about the time of the onset of this condition.

Venereal History.—He denies luetic infection; a year ago he had a gonorrheal infection with swollen glands.

Before his present illness he would drink a good deal, up to a quart of whisky in a day.

By occupation he is a polisher in a piano factory; why is that important?

INTERN: Because it might be a metallic poisoning.

DR. HAMBURGER: Yes; a polisher in a piano factory or a polisher of brass or other metal is very likely to develop metallic poisoning. (Addressing Patient): How long have you been a polisher?

PATIENT: About four years.

DR. HAMBURGER: You did no polishing in your own country?

PATIENT: No, I was a miner for a while.

DR. HAMBURGER: He apparently had his complaint before he was a polisher. His blood examination showed hemoglobin 70 per cent.; 9200 whites. The differential count showed 20 per cent. small mononuclears, 4 per cent. polymorphonuclears, 76 per cent. neutrophils. A single examination of the urine was negative; a twenty-four-hour specimen showed a specific gravity of 1020. He was given an Ewald meal, consisting of two slices of toast and two cups of tea, and at the end of one hour showed a free acidity of 20, and a total acidity of 70; also a positive Weber. On the following day that was repeated, and the free acidity showed 20 and the total 30, with a negative Weber. A stool examination showed a positive Weber on the 13th, on the 14th a negative Weber. He has had no temperature at all; his pulse has been regular, running between 68 and 74, respirations 18 to 20.

Yesterday morning we fluoroscoped him. He was given the regular motor bismuth meal to test the motility of the stomach. Six hours later the stomach was completely empty, showing a normal emptying time. The second bismuth meal examination by Dr. Murphy showed the outline of a normal, orthotonic stomach, the bismuth appearing very promptly in the duodenum, active peristalsis, and no filling defects. In other words, the radiographic examination of the stomach and duodenum is negative.

Now, on examining him you see a young, vigorous, robust laborer, sitting up quietly in bed, not apparently acutely ill, good color, some slight loss in weight, but, on the whole, a man not apparently seriously ill. His skin is warm and moist, his pulse is full, regular, and slow. His skin is clear; there is no jaundice and no cyanosis. He has on the upper portion of the left chest a somewhat unusual reddish-brown maculopapular rash; otherwise his skin is negative. This rash is somewhat suggestive of a tertiary syphilitic skin lesion, although it likewise has some of the characteristics of a *tinea versicolor*.

Going over him very hastily, his pupils are equal and react promptly to light and to accommodation; his teeth are in remark-

ably good condition; there is no pyorrhea. He has no lead-line which might be present from his being a polisher.

(Addressing Patient): Have you had sore throat?

PATIENT: Yes, sometimes.

DR. HAMBURGER: Did you, seven years ago, when this trouble started?

PATIENT: Sometimes.

DR. HAMBURGER: His tongue is slightly coated and shows a very faint tremor. He has rather large tonsils, particularly the left one, which shows evidences of old infections. The whole pharynx is a bit red. (Addressing Patient): Do you smoke a good deal?

PATIENT: Yes, I smoke all the time; I started when I was twenty-four.

DR. HAMBURGER: Do you smoke cigarettes?

PATIENT: Yes, but I do not chew.

DR. HAMBURGER: He has no cervical adenopathy. His chest is well developed and the musculature is good. The respirations are good, free, and symmetric. On percussion, the liver dulness begins at the lower border of the fifth rib, and flatness at the lower border of the sixth. The excursion is good. The chest resonance is normal throughout; no hyperresonance. His heart borders are apparently within normal limits. On auscultation, there is nothing abnormal; the tones are regular and clear. The only striking thing is a somewhat increased second aortic tone. That is interesting in a young man with a systolic blood-pressure of 130. There is a ringing metallic character to the second aortic.

His spine is perfectly straight, no curvature in any direction. There is a practically free excursion, although probably some very slight restriction on the right low down. There is good respiratory excursion; on auscultation there is nothing abnormal.

On inspection of the abdomen (and here I may say that the best way to look at an abdomen is with the feet toward the light) we find it symmetric and the development of the recti muscles very plain; there is no evidence of visible peristalsis, either above or below the umbilicus. The tissue about the umbilicus is probably just a bit more prominent than normal. There is a very

slight umbilical hernia and he complains of tenderness on pressure. On palpation of the abdomen you feel very distinctly the sigmoid roll under your finger like a sausage-shaped mass, and there are also some small bodies about it. On palpating near the median line he says it is sore. He complains of tenderness on pressure all around the umbilicus; this tenderness apparently is variable and the pain apparently radiates toward the lower portion of the abdomen. The abdomen is sensitive, particularly below the umbilicus. On palpating for his spleen it cannot be felt, and on percussion the splenic dulness is not enlarged, so it is probably safe to conclude that the spleen is normal. The liver is not palpable, there is no increased resistance, and we are justified in concluding that the liver is not enlarged. Deep pressure over the appendix causes some tenderness. On attempting to reinforce the findings, by stretching the iliopsoas muscle, by having him attempt to raise his right leg against resistance, and then pressing on the appendix region it increases this tenderness somewhat; on the left he also seems to have more tenderness on stretching the left iliopsoas.

On percussion, we find the greater curvature of the stomach just above the umbilicus; the lesser just below the xiphoid. He has a general adenopathy of his inguinal glands and of the epitrochlears.

On examination of his legs the soft tissues show no pitting and there is the same good development of his muscles. The knee reflexes are brisk and equal; there is no Babinski; superficial and scrotal reflexes are present and intact; abdominal reflexes equal and present.

To sum up the physical examination. The only findings that are of interest are:

- (1) His accentuated second aortic tone.
- (2) The slight general adenopathy.
- (3) The small shotty glands in the neck.
- (4) The tenderness about the umbilicus and in the right lower quadrant.
- (5) There is the suggestion of loss of weight (the difference between his skin and subcutaneous tissue).

(6) The heart and lungs are negative.

(7) There is a slight maculopapular eruption on his skin.

On going over him the other day I thought that from the presence of this rash and the localization of the glands a Wassermann should be made, although a diagnosis of a gastric ulcer had been made and he had had only a gonorrheal infection, and the report on the blood comes back strongly positive.

We will now distend his stomach by giving him 1 dram of soda bicarbonate and $\frac{1}{2}$ dram of tartaric acid in water, having him drink the acid first. This is a valuable test for bringing out the stomach outline and of causing visible peristalsis. With the stomach distended he complains of no pain at all, and this is interesting, because in true ulcer of the stomach there is often increased pain because of distention. Although the stomach is distended he has no pain at this time, although he probably should have been given a larger amount, for he has a large stomach.

Now what is the matter with him? A man complaining of stomach distress for seven years, principally of pain coming on after meals, with relief by food and by induced vomiting, and with a loss of weight of 40 pounds; tenderness about the umbilicus, a positive Wassermann, a general adenopathy, and a skin eruption.

Before attempting to arrive at a definite diagnosis let us talk for just a minute about abdominal pain. In some nervous individuals a normal physiologic sensation may be called *pain*, while in a more phlegmatic person it will not be noticed at all. A highly nervous woman will interpret a normal peristalsis as pain, while a more stable individual will recognize the sensation as normal. We might classify it something like this:

- | | | |
|------|---|--|
| Pain | { | (1) Any sensation. |
| | | (2) Abdominal distress or discomfort. |
| | | (a) Sensation of fullness, when present. |
| | | (b) Pain, in different individuals. |
| | | (c) May be sensation of weight or pressure. |
| | | (d) May be sensation of nausea or indigestion. |
| | | (e) It may be true pain. |

Secondly, one should distinguish clearly between subjective pain and tenderness; the latter—tenderness or pain on pressure—

is an objective finding, either found by the patient or by the physician on examination. This morning I want to speak particularly of the former, viz., spontaneous pain.

Another differentiation to be made in the consideration of pain and the diagnostic importance of pain is whether it is, first of all, a constant and persistent pain, or whether the pain is intermittent, periodic, or anginoid; whether it is present all the time or whether it comes and goes, has daily variations, seasonal variations, or is cramp-like or colicky. When a patient comes in complaining of cramps he means the intermittent, periodic pain. In the majority of cases that come in complaining of abdominal pain it is the periodic pain that is complained of. Probably 95 per cent. of all patients complaining of abdominal pain have pain of this variety. In any patient complaining of intermittent pain, such as this individual, before you can possibly start to connect the history of pain with disease of any organ or group of organs you must get as much information as possible about the pain itself.

To do this one must obtain as accurate and detailed a history as possible, and in this regard experience and training is of greatest importance. One may develop a technic of obtaining an accurate and detailed gastric history the same as one may develop his technic of doing an appendectomy. Furthermore, the accuracy of a pathologic diagnosis bears a direct relationship to the accuracy of the clinical history. In endeavoring to obtain an accurate history of this type of recurring abdominal pain one must inquire first as to the localization of the pain and, particularly, whether it is *diffuse* or *localized*. Secondly, one inquires whether the pain remains constantly in the region indicated, or whether it radiates out from here into other regions, and, if it radiates, in which direction does the pain travel. In this way one inquires, for example, whether there is radiation toward the shoulder regions, whether there is radiation toward the back, and if toward the back, does it go around the sides of the body or directly through; whether there is radiation toward the lower portion of the abdomen, toward the bladder, or toward the extremities. Third, the time of the occurrence of the pain. Inasmuch as the

pain that we are considering is of an intermittent character, it must, of necessity, have periods of recurrence. Is the pain more prominent day or night? Do the pains have relationship to any physiologic act? Do they bear relation to eating, to swallowing, to bowel movements, to urination? Do they come on with change of posture? If the pain has a relation to eating, we can with profit trace the entire twenty-four-hour period accurately. For example, taking the patient upon waking in the morning, does he have pain before he gets up, or does the pain occur upon getting out of bed? Does he have pain before breakfast? Does it occur after breakfast, and at what time after breakfast? If it occurs after breakfast, does it last until the next meal or does it disappear spontaneously? What influence has the noon meal on the pain? Does the recurrence of the pain after the noon meal correspond with the occurrence after breakfast? Does it last until the evening meal? At what time does the pain occur after supper? Does it last until bedtime? Does it prevent sleep? If it disappears before sleep, does it wake the patient up during the night? If so, at what time? Or is the patient's rest undisturbed?

As has been said, such an accurate history is only possible with an intelligent patient, but an attempt should be made with *every* patient to obtain as accurate a history in this regard as possible. In this connection, Lockwood has devised a rather unique chart for the graphic representation of pain:

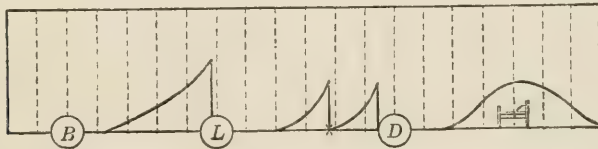


Fig. 92.—Classical ulcer pain. The vertical dotted lines indicate hours. The three circles, B, L, and D, indicate breakfast, lunch, and dinner. The time of retiring is indicated by the outline of the bed. The mark x in this chart indicates extra nourishment. (From Lockwood, "Diseases of the Stomach.")

He recommends that in various chronic conditions of the stomach that such pain and distress records be continued for a

full week. The following chart shows such a record taken from his text, "Diseases of the Stomach":

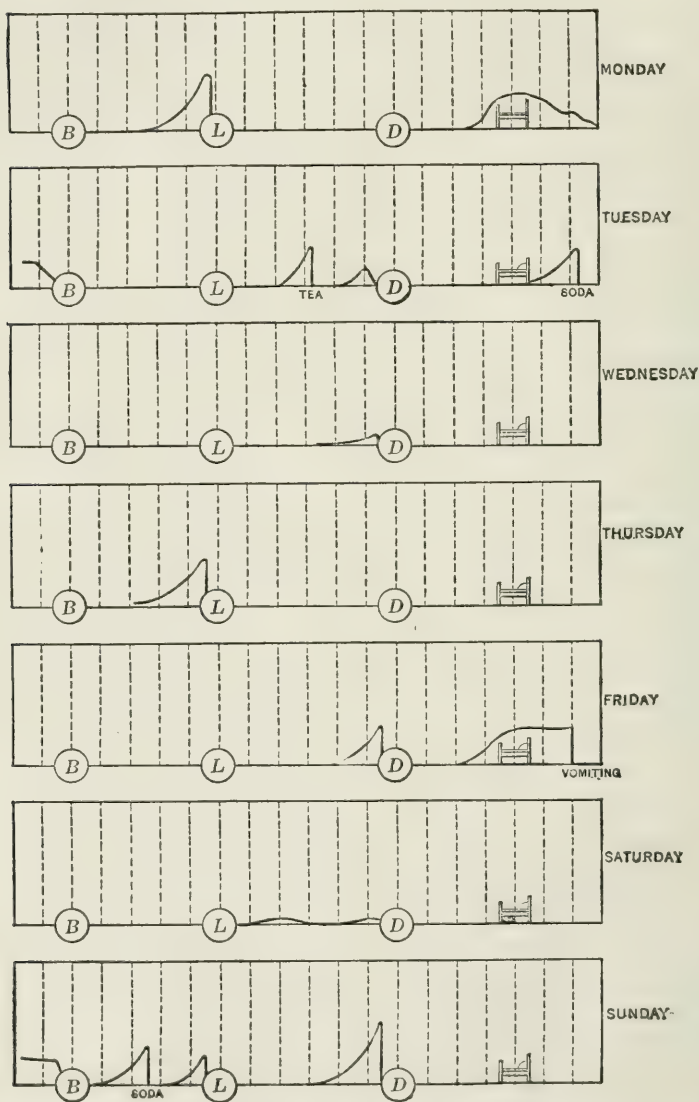


Fig. 93.—Pain chart of hypersecretion from chronic appendicitis. (From Lockwood, "Diseases of the Stomach.")

Fourth, the nature of the pain itself. Does the patient actually complain of true pain, or is it merely distress? The importance of stomach "distress" is so great that it must be left for discussion at another time. We can only emphasize here that, in all probability, many if not the majority of gastric complaints, which are of the nature of "distress" and not true pain, are simply lesser gradations of the latter; that is, in a long-continued stomach disease the earliest manifestations are such complaints as fulness, pressure, and indigestion coming on after meals, and only later, as the disease progresses, do these amount to true pain. And further, as has been said, a complaint which in one patient may be described as merely "distress," in another, more highly nervous patient, will be called a true pain. If, however, to revert to our original discussion, the complaint is a pain itself, what is the character of this pain? Is it a dull ache or is it a sharp, cutting, sticking pain? Is it a mild, benign pain, or is it an intense, agonizing pain? Is the pain of a burning, gnawing, or hunger character?

Fifth, pain relief. What relieves the pain? Is it relieved by further eating? By hot foods or cold foods, by liquids or solids, by a large or small meal? Is it relieved by a bowel movement? Does rest in a recumbent posture give relief, or is it made worse? Does exercise or activity give relief or make it worse? Is it relieved by vomiting either spontaneous or induced? Is it relieved by pressure to the stomach or heat or cold applied to the abdomen? Is it relieved by an abdominal binder or other abdominal support? Is it relieved by assuming any special posture, right or left side, by the knee-chest position, by elevation of the foot or the head of the bed?

Sixth, the associations of pain. What other subjective complaints have been noted? Is there associated nausea or indigestion, and, if so, does the nausea or indigestion precede or follow the pain? Is it associated with vomiting? Is there associated pain in other regions of the body, such as headache or chest pain, or pain in the extremities? Is it associated with fever or with chills, or with sweating, or with a rapid pulse, or with cardiac palpitation? Is it associated with cyanosis, dyspnea, pallor, or icterus?

Summary of Gastro-intestinal History—Test Meal Case.			
Office No. A.....	Name.....	Date.....	
Age.....	Physician.....		
Previous History.....			
Comes on Account.....			
Duration of Disease.....			
Mode of Onset.....			
Present Complaint.....			
WEIGHT—Present.....6 mo. ago.....1 yr. ago.....5 yrs. ago.....			
Disturbances in swallowing.....			
ABDOMINAL DISTRESS OR PAIN.....			
Where.....			
When.....			
Kind.....			
Duration.....			
Transmission.....			
Relation to food and drink.....			
Relieved by.....			
Relation to body movement.....			
VOMITING..... Character.....			
Frequency.....			
Brought on by.....			
Time of day.....Delayed Vomit.....			
Relieved by.....Vomit Relief.....			
Nausea.....When?.....			
ERUCTATIONS, BELCHING, REGURGITATION, WATER BRASH.....			
Time.....			
Frequency.....			
Taste.....			
Relieved by.....			
Appetite.....			
BLEEDING.....			
Bowels.....Flatulence.....			
REMARKS.....			
.....M. D.			

Fig. 94.—History card.

In this way one could continue almost indefinitely, and it is manifestly quite impossible completely to cover this entire sub-

Report of Gastric Examination.	
Office No. A.....	Age.....Physician
Name.....	Address.....
PASSAGE OF TUBE.....	
ABDOMINAL EXAMINATION.....	
INFLATION OF STOMACH.....	
Bulbs.....	G. C.....F. B.....Navel
GASTRIC EXTRACT.	
1. Meal	Time.....Lavage water used.....
MACROSCOPIC EXAMINATION.	
Chymification.....	Color.....Odor.....
QUANTITY.....	Total.....Filtrate.....
FOOD REMNANTS.....	Blood.....Bile.....Mucus.....
Tissue Bits.....	Foreign Bodies.....
Remarks	
2. CHEMICAL EXAMINATION.	
TOTAL ACIDITY.....	Method
Altered Blood.....	Free HCL.....Combined HCL.....
Special Tests.....	Lactic Acid.....Fatty Acid.....Bile.....
3. MICROSCOPIC EXAMINATION.	
Food Remains.....	Method
Starch Digestion.....	
R. B. C.....	W. B. C.....Yeasts.....
Sarcines	Oppler Boas Bacilli.....
Bacteria or Protozoa.....	
EXAMINATION OF STOOL.....	
RECTAL and Sigmoidoscopic Examination.....	
RADIOGRAPHIC EXAMINATION.....	
EXAMINED.....19.... by	
DIAGNOSIS	
TREATMENT.....	

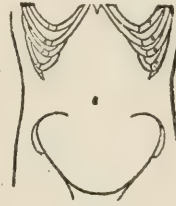


Fig. 95.—History card.

ject of obtaining an accurate stomach history. If you are particularly interested in this phase it would be profitable to read

Boas, *Diagnostik und Therapie der Magenkrankheiten*, in the introduction of which book he devotes 16 pages to a discussion of history taking in gastric disease. We have in use in the Dispensary a fairly complete history card for obtaining an accurate history which may be of some interest.

Returning to the discussion of this patient, and beginning to attempt to answer the question, What is the matter with him? we might begin the discussion of this question by attempting to determine what relationship exists, if any, between his physical findings, particularly the finding of abdominal tenderness, and the patient's complaints. It should be said that this complaint of pain after meals is one of the most common forms of intermittent pain. What is the cause of pain coming on after meals? First of all, we might divide these pains into early pains, those coming on during the meal or immediately after it; and, second, late pains, those coming on thirty minutes to one or two hours afterward. What are the causes of the early pains? Those coming on during the meal or immediately after it may be associated with deglutition and be due to disease of the esophagus, such as carcinoma, stricture, diverticula, cardiospasm, etc., and in this patient such a suggestion arises because of the pain coming on immediately after eating. It is very often a so-called neurosis. In neurotic individuals with neurotic pain elsewhere you often get a pain immediately upon taking food, and that pain is probably nothing more than the food entering the stomach, which is highly sensitized, and such people call it pain.

Pain immediately following eating suggests a neurosis, and is common in neurotic individuals, as emphasized by Cohnheim, of Berlin. It might be an ulcer of the cardia with cardiospasm and caused by the irritation of the food. It may be due to adhesions; they will give early pain. Dr. Friedman has recently analyzed a number of such cases and found where the pains were most definite, coming on early, that there were often adhesions connecting the curvatures of the stomach with the gall-bladder or elsewhere, and he has shown that frequently the pains coming on from fifteen minutes to half an hour after meals are adhesion pains.

Now about the late pains, those coming on one to four hours after eating, the so-called ulcer pain. This man has that type of pain; has he, therefore, an ulcer? If he has an ulcer, why does he have these late pains? We will speak just for a minute about these late pains. It is obvious that pain coming on in this way can be due to changes in the acid content of the stomach; that is, *to changes in the acid secretion of the stomach*. If it is due to a secretory phenomenon, why should it come on half an hour to two hours after meals? It has been said that this late pain, coming on one-half hour to two or four hours after eating, is due to the increased concentration of gastric acidity. In attempting to answer this question for ourselves we have been passing Rehfuß tubes into the stomachs of gastric patients, and aspirating the stomach contents at intervals of ten or fifteen minutes. And apparently, in many instances, the most severe complaint of pain is associated with the highest degree of free acidity. However, there are many exceptions to this relationship, and these will be discussed later. While these late pains, as has been said, may be due to changes in the acid secretion of the stomach, it is also possible that they may be due to changes in *gastric motility* or, possibly better said, changes in the *tone of gastric muscle*. And in this regard it is apparent, particularly to those of you who have watched under the fluoroscope the peristalsis of the stomach following the ingestion of bismuth meals, that approximately one-half to one hour after the meal peristalsis is at its height and that gastric motility is at its height. In other words, one must rule out these changes in peristalsis and muscle tone, occurring at approximately the same time as the increased acidity, to decide the direct etiologic relationship of the pain; that is, it is conceivable that the subjective complaint of pain may be due to either changes in gastric secretion or in gastric motility, or both.

Is it a motor phenomenon due to increased peristalsis? The causation of this type of pain still is a question in the minds of a good many men and they are still divided about it. The current belief is that the pain is due to the corrosive free acid acting on the ulcer, and an analogy has been drawn to the effect that free HCl

of 0.3 per cent. dropped on the skin will smart and burn. However, there is good evidence by Hertz, of London, that this is not the case. In "The Sensibility of the Alimentary Canal" he has demonstrated that 4 to 6 ounces of a 0.5 per cent. hydrochloric acid introduced into the stomach which was proved to have gastric ulcer caused no pain. I have repeated this observation on several occasions, and have never been able definitely to prove the relationship between the introduction of free hydrochloric acid in the stomach of an ulcer patient and the subjective complaint of pain. This is pretty good evidence that increased acidity does not cause pain. You will also remember that Prof. Carlson said that when the acid accumulates in the stomach of Mr. V. he complains of no pain and no discomfort. Likewise, Hertz and others have shown that the mucous membrane of the stomach is insensitive to most stimuli which will produce sensations, both tactile and thermal. They will not produce pain in a normal stomach, and that, to my mind, is good evidence that the pain is not connected with the secretory change. Hertz, again, has shown that if you distend the stomach greatly with gas or by pumping in air with a balloon, the first complaint is of fulness, and then the patient complains of pain, and Hertz concludes that the pain is due to the tension producing stimuli on the muscle wall of the gastro-intestinal tract. Hertz believes that anything which will produce tension will produce pain, and he reasons out that, first of all, the chemical contents of the stomach have no or little sensory effect one hour after eating, the peristalsis is at its highest, and in individuals where you get late pain there is increased peristalsis, and this is due to three factors: First of all, the hypertonic contraction; second, a prolonged closure of the pylorus, called "pylorospasm"; and, third, increased peristalsis. The peristalsis forces food against the walls of the stomach repeatedly and causes pain.

Such increased peristalsis and pyloric spasm, plus an increased acidity or hypersecretion, has been called by many writers the "pyloric syndrome." The pyloric spasm, plus increased peristalsis, plus increased acidity, is the pyloric syndrome. Now why does soda relieve it? It certainly does relieve it, and

the explanation is probably something like this: Injecting acid into the stomach causes increased peristalsis, but if you neutralize the acid the peristalsis diminishes and the pain disappears on the relaxation of the pyloric cramp.

INTERN: The acidity is decreased at the same time, and it seems to me it is hard to say which is causing the relief.

DR. HAMBURGER: Not if you accept the explanation that the pain is due to the peristalsis and not to free HCl. The importance of this differentiation is that, while the pyloric syndrome does occur in ulcer, it occurs in a whole variety of conditions other than ulcer, and the broader our experience is, the more we are convinced that it is simply an expression of increased muscle tone. For example, think of conditions in other organs. You get this pyloric syndrome in neurotics who have been operated on time and time again for pyloric and duodenal ulcer without finding any trace of ulcer. It occurs in anemia with a hypersensitive nervous system. It occurs in pulmonary tuberculosis. In many cases where this late pain with hypersecretion and pyloric spasm have served as indications for laparotomy, exploratory laparotomy has failed to reveal an ulcer, but has revealed diseases of other abdominal organs; for example, the gall-bladder, the appendix, the colon, the pancreas, the liver, the spleen. In such cases the absence of a raw spot in the stomach or duodenum makes one look for other causes for these late pains, and evidence such as this lends additional support to the belief that these late pains are due to changes in muscle tone and not to irritation of the base of a chronic ulcer.

We have had several cases in Michael Reese Hospital, sent in as gastric and duodenal ulcer, which have proved to have a perfectly intact gastro-intestinal tract, but examination of the lungs revealed a tubercular lesion, and the pain disappears on treatment for the lung condition. In one case we had a tailor; his wife said, "When he gets this pain his lips get blue." That suggested that it might be a circulatory pain, and we discovered that he had an early cardiac insufficiency. Treatment, consisting of rest in bed and digitalis, resulted in the pains disappearing. He had the pyloric syndrome, which was diagnosed and treated as ulcer, but

it disappeared on treatment for his heart condition. In chronic appendicitis you have the same thing, and if you take out the appendix the pain will disappear. I have also seen a man who had a slight pain over his appendix, the same as this man has, and a surgeon removed the appendix, and six months later the patient developed a pyloric syndrome. If you examine these cases carefully and accurately you will find that they have adhesions, and the reflex set up by the adhesions about the cecum produces a spasm. You cannot do very much for a case like that. Patients are permanently invalidated by a thing of that kind. Primarily, they have nothing wrong but a hypersensitive, nervous stomach.

I want to get firmly entrenched in your minds that the so-called "ulcer history" is often due to something entirely different than an ulcer. I cannot mention them all. Very often the first symptom of Bright's disease is this pyloric syndrome. Go over these patients carefully and you will find a high blood-pressure and all the other symptoms. Ulcer causes the same thing. Why? Because you have a raw spot, and this causes increased acidity and constantly keeps up and increases the pyloric spasm.

There is one other point that I want to speak of, viz., the nerve ends in the raw spot or the ulcer. What does hydrochloric acid do to the raw spot? It probably stimulates these nerve ends to induce hyperperistalsis and possibly an increased activity, and the pyloric syndrome is due to the action of the acid on the raw spot, not by causing the pain, but by stimulating the nerve ends and stimulating hypertonicity.

The management of these cases we will have to leave until some other time.

To speak just for one moment about this patient: I believe that he has no ulcer. He has a slightly increased second aortic tone, has adenopathy, and has a skin lesion and a strongly marked positive Wassermann. We will put him on antisyphilitic treatment. He has no positive evidence of syphilis except the double plus Wassermann, but it is a big question whether the syphilis has any relation to the gastric pain. We will put him on antisyphilitic treatment and see if the pain will not all clear up.

INTERN: If he has ulcer, doesn't the pain come on pretty early?

DR. HAMBURGER: Yes; but that does not exclude ulcer, for you may get many different and bizarre clinical pictures in ulcer. We are going to try to find out why he has this fifteen-minute pain. He is probably a neurotic, and has a hypersensitive nervous stomach with a hypertonus of the muscularis, and that gives him pain.

To sum up, then, these late pains, or so-called "ulcer pains," occur in chronic ulcer of the stomach. They likewise occur, however, with equal if not greater frequency in conditions other than ulcer. And these conditions are legion. Therefore, before one can accurately manage or treat conditions of periodic abdominal pain, so-called ulcer pains, one must rule out by careful examination and by careful history all these varied conditions, or must find positive direct evidence by examination and history of chronic ulcer. Probably the most frequent explanation of the failure, both medical and surgical, to cure "ulcer" of the stomach is because there is no ulcer; the treatment having been directed to the so-called "ulcer" history and not to the actual disease conditions present.

At another time I hope to take up the question of the direct diagnosis of chronic ulcer, but I would merely emphasize here that intermittent abdominal pain may be due to a variety of conditions and that these conditions must all be differentiated, and that the beginning of such differentiation lies entirely in the taking of an accurate clinical history.

September 25, 1915.

You will remember that we went over the discussion of the history and findings of this patient's case last week, and concluded that in all probability he had a tertiary syphilis, and that the gastric findings were probably due to a group of symptoms known as the pyloric syndrome.

The question comes up as to medical management,—what to do in a case of this kind coming in for advice and treatment,—and it should be said that cases of what we call the pyloric syndrome

usually respond well to properly controlled medical treatment. However, if treatment is not accurately directed and properly controlled, they very quickly lose faith in medical management and seek out a surgeon who can be prevailed upon to open them up. The operation usually results in negative findings surgically, and the patient either has the same complaints or increased complaints due to adhesions, and he becomes a chronic case. So it is up to the medical man to outline a treatment and so to manage it that it will be satisfactory.

First of all, if this man has a tertiary syphilis, where is it? It is pretty difficult to say in his case. The only definite findings are the general adenopathy and the increased aortic tone, so that as far as one can see from the physical examination the only definite involvement is that of the lymph-glands and probably a very early aortitis with increased second tone. But, in any event, he has a positive Wassermann, and ought to be given the benefit of antisyphilitic treatment. It is probable that if he has a tertiary syphilis the whole gastro-intestinal trouble is due to this, and the whole thing will probably disappear if the antisyphilitic treatment is vigorously and actively carried out.

This patient should have mercury rubs, a gram a day for six days, on alternate parts of the body; first the arms, then the abdomen, then the thighs and buttocks, and then the legs, with a cleansing bath on the seventh day, and then starting all over. Give him twenty or more rubs. In addition, iodid of potassium, increased 1 drop daily to tolerance, and during the administration of mercury and iodid special attention should be given the kidneys, owing to the danger of setting up a transitory nephritis, and the eyes should be watched carefully on account of the possibility of setting up an iritis. Also the use of a potassium chlorate mouth-wash to control possible salivation.

In cases of gastric complaints of tertiary syphilis that do not quiet down with antisyphilitic treatment, what do we do for the stomach symptoms? In attempting to manage medically a case of this kind you want to keep clearly in mind what you are attempting to manage, and, as said last week, it is our belief that in patients complaining of abdominal pain the pain is not due

to the stimulation of the raw spot by the hydrochloric acid, but is due to the increased peristalsis and pylorospasm. Therefore the management of these cases narrows itself down to the treatment of the increased tone and peristalsis, and resolves itself down to the control of the free acid as well as the tone and peristalsis.

What measures have we as medical men with which to control the increased motility and peristalsis? They may be grouped: First, dietary measures. Second, medicinal measures. Inasmuch as the treatment of a group of symptoms such as these—that is, pyloric spasm, hypersecretion, and resulting pain—is very largely the syndrome that you get in chronic ulcer, carcinoma, and in certain forms of gastritis, the management of this syndrome may be said to be the management of ulcer and most of the medical management of stomach cases, so that we shall go into considerable detail as to these different measures.

This morning I propose to begin the discussion of some of the questions of diet. I don't propose to attempt to cover the whole subject, even of diet, but simply to make a start on certain dietary principles and their effect upon motility and secretion. Included in these various clinical talks we will take up discussions of other units of medical management.

I think it may be advisable to begin the discussion with the effect and value of the use of water, because water is one of the simplest, commonest, and most universally used parts of the diet, probably the simplest, and we have been using water here as a method of testing out this case, and I thought it might be well to have a discussion on the effect of water in such cases.

There are just a few things I want to say about it. Heidenhain, as far back as 1888, was probably the first man to show that water had a stimulating effect on gastric secretion, and since then there has been a lot of experimental work done, especially during the past three years, on the effect of water on the gastric secretion in man. Experimenting on dogs, Pawlow and others, most recently Carlson, have shown that water is a true gastric stimulant. There has been considerable work as to the mechanism of this water stimulation. For instance, a Russian, Krschysch-

kowski, in 1906 showed that water as well as meat and salt and all of the food-stuffs that go to make up a meal are without secretory effect upon the *fundus* of the dog's stomach; that is, with the stomach separated into two sections the introduction of these foods had practically no secretory effect upon the stomach fundus itself. There are certain points that had to be taken into consideration in the use of meats, meat extract, etc. If the animal did not see or smell the food, the mere introduction of the food-stuffs was without secretory effect upon the stomach. We might also note that water introduced as an enema causes no secretion, just as soda introduced as an enema inhibits secretion and alcohol stimulates secretion.

In 1894 another Russian, working with Pawlow, showed that 200 c.c. of water introduced into the large stomach caused a definite secretion in the small stomach, a secretion which contains pepsin.

Two years later Lobassow showed that with a patent pylorus the water leaves the stomach quickly. Then, in 1913, the point was proved up pretty definitely by Sawitsch and Zelgony, who showed that water in the pylorus caused a definite secretion, and showed further that water in the duodenum caused very little secretion.

I might say that as far as we can conclude from the work of the men just mentioned the introduction of food-stuffs into the fundus of the stomach is without secretory effect, and it has been shown further that, while there is very slight absorption of water from the stomach, in general there is practically no absorption from the fundus end and very little from the pyloric end.

In the work on normal young men Bergeim and Hawk found some interesting things. They found that water, either introduced by stomach-tube or simply swallowed by the patient, leaves the stomach very rapidly, probably by means of a trough along the lesser curvature. This trough is called by various names, by some the "canalis gastricus." It is pretty well accepted by all authors now that the water leaves the stomach very promptly by means of this trough along the lesser curvature. Holzknecht

showed that it took sixteen minutes for 200 c.c. to leave the stomach, and Dr. Carlson thought that it took seventeen minutes, but both found that this amount of water left the stomach in from ten to twenty minutes, so we can say that water leaves the stomach very rapidly.

In our work here we propose to test the time that the water and other food-stuffs leave the stomach by means of coloring-matter, such as carmin and chlorophyl, and in the work we have done so far we find that 200 c.c. leaves the stomach in from thirty to forty-five minutes, and that is about in accord with Carlson and Bergeim and Hawk. The time is variable in different stomachs, but it averages about thirty to forty-five minutes.

So far as the secretion of gastric juice is concerned, we have found that as small an amount as 50 c.c. of water produces a marked secretion of gastric juice. You may remember that Sokolow in experiments on animals found there was a latent period, but in our work and in that of Bergeim and Hawk there is no latent period. The free acid begins to rise immediately, with no latent period, so we conclude that water is a strong stimulant, and in some instances yields an acidity as high as 100.

There are certain conclusions that can be drawn from this in the medical management of these cases. For instance, the fact that water is a strong gastric stimulant is of interest, for, if you are trying entirely to keep down the secretion of the stomach, water would have to be prohibited because it is as strong a stimulant as the average Ewald test-meal. Experiments have shown, for instance, that the acidity is greater after water than after a test-meal, so the control of the water stimulant must be carefully watched.

As far as the motility is concerned, we say the stomach empties itself rapidly of water, but, although the water itself leaves the stomach rapidly, there is left the stimulation of the gastric secretion. You will remember the curves we have had here; the difference in the curve in swallowing 100 c.c. of distilled water, and the difference in response in swallowing 400 c.c. of distilled water. It was shown that within five minutes in 100 c.c. the acid started the rise, and in 400 c.c. it took thirty minutes to rise. So this

must be considered in mapping out a diet, and it might be said, in addition, that water, of course, is an absolute necessity in the life of a person and must be introduced into the body in some way, so that we must arrive at some means of treatment to overcome the effect of water stimulation. One way of controlling water stimulation is to give it per rectum, where it causes little or no secretion. Water by mouth, however, cannot be withheld long.

We come down to the medical management. Perhaps the three general classes of drugs which can be used to overcome water stimulation are, first, the alkalies; these being used in two ways, to reduce the acidity and to inhibit the gastric juice; second, oil or fat, which will decrease the gastric juice secretion; and, third, atropin and belladonna.

I propose during these clinics to take up the discussion of milk, cream, certain fluids, such as broths of various kinds, meat extracts of various kinds, and the effects of fruit juices on the gastric secretion, and then the effect of meat stuffs, fowl, the effect of sugar, and then the similarity between the effects of fluids and solids, a dry diet and a moist diet.

There is much more to be said about the effects of water, but I think we have probably covered the principal facts. The water surely leaves the stomach very quickly, certainly within forty-five minutes, and the secretion obtained after that is gastric juice, and the use of coloring-matter is a good way of indicating the disappearance of water through the stomach. There is one thing more. If we accept the theory that the disappearing place for the water is the pyloric end, it is probable that with an obstruction of the pylorus the retention of water will keep up the stimulation of gastric juice, and that causes the pyloric cramp; so that the rapid exit of water through the pylorus brings relief. If you have good motility you should only get a minimum secretion.

NOTE.—The patient was put on mercury inunctions and potassium iodid in increasing doses and the full ward diet, and his abdominal distress has almost entirely disappeared. He has gained in weight, and shows every prospect of complete relief and early discharge from the hospital.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

SUBACUTE LANDRY'S PARALYSIS

DR. MIX: The case of the patient we are to see is very different from the usual run of cases of disease of the nervous system. The patient is a boy, seventeen years of age, who by occupation is a farmer from the Canadian northwest. He entered the hospital on the 20th day of February, 1915, with the following history: Family history is entirely negative; his parents living and well; brothers and sisters all well. (To Patient): No deaths in the family? No tuberculosis in the family?

PATIENT: No, sir.

DR. MIX: His only previous illness was measles at the age of thirteen, and that was a very mild case, because he was sick only a week. On the 18th of January, that is to say, two months ago, lacking three days, he went out driving in a cutter. It was pretty cold. (To Patient): How cold was it, Ross?

PATIENT: It was about 10 degrees below zero.

DR. MIX: And how far had you been riding?

PATIENT: Two and a half miles.

DR. MIX: And then he noticed he was numb in his feet below the ankles; that may have been simply a chilling of the feet. His feet may have gotten cold, but in the light of what subsequently happened it may have more meaning than just simply cold feet. (To Patient): When did you go skating?

PATIENT: One-half hour after.

DR. MIX: How long did you skate?

PATIENT: Two hours.

DR. MIX: What time did you get home?

PATIENT: Eight o'clock in the evening.

DR. MIX: And then he noticed that the numbness was perhaps a little greater in his feet. He went to bed and slept all right, but the numbness remained in his feet and it was there the second day after. On the third day the numbness began to rise, and by the third day it had ascended from the feet to above the ankles. After that the numbness went each day a little bit higher. *It was not associated with pain at all.* Please underscore that there was no pain at the onset of this difficulty.

Twelve days later the numbness had ascended to above the knees and he then began to have difficulty in walking. At each step the legs would cross; that is, the legs came one in front of the other and he would stumble. The numbness remained just about the same in degree, and no pain appeared, to February 1st. About the 9th of February, that is to say, eight days afterward, he began to have difficulty in walking; his knees gave way entirely, so that he was unable to stand upon his feet. He developed a paraplegia and he was compelled to take to his bed. About the time that the knees gave way so that he could not walk he began to have some pain in the calves of his legs and in the knees.

On the 9th of February *pain* appeared and increased in the calves and in the knees. (To Patient): The pain was never very great, was it?

PATIENT: No.

DR. MIX: It did not make you cry out and you never had treatment for it; it just bothered you?

PATIENT: Yes.

DR. MIX: In other words, the pain was not the severe lancinating pain that is usually caused by pressure upon or inflammation of the posterior sensory roots. The pain remained about the same until the present time, but perhaps a little more marked at night.

About the time of complete loss of use of the legs he also had pain in his right armpit; that is, right down here (indicating armpit) and coming across the chest. There was some pain upon pressure across the chest, but in three days this pain entirely disappeared. About the 6th of February he began to have difficulty in urinating. It was possible to empty the bladder only twice a

day. Never did he have involuntaries. But a week before entering, which is the 13th of February, the bowels quit moving, except by the use of an enema. (To Patient): Since you have been in the hospital you have no difficulty in emptying the bladder?

PATIENT: A little bit.

DR. MIX: What kind of difficulty?

PATIENT: In starting it.

DR. MIX: That is, you can't relax?

PATIENT: Yes.

DR. MIX: Evidently a slight loss of control of the sphincter of the bladder. (To Patient): How has it been with the rectum since you have been in the hospital? Have you had difficulty in emptying the rectum?

PATIENT: No.

DR. MIX: Now, since he has been in the hospital he has complained a good deal of headache and he has not infrequently had some vomiting. (To Patient): You vomited yesterday?

PATIENT: Yes, sir.

DR. MIX: Did you vomit today?

PATIENT: No.

DR. MIX: Yesterday morning?

PATIENT: Yes, sir.

DR. MIX: And you have a headache now and then?

PATIENT: Yes, sir.

DR. MIX: More than you had a while ago?

PATIENT: Yes, sir.

DR. MIX: That is the history. The history is that of some sort of a process which is acute or subacute.

Now when we come to examine the history a little further one thing is rather evident, that is, *there is no great amount of pain*. The reason I speak of that is this: This patient was sent to the hospital with the diagnosis of a tumor of the spinal cord. A tumor usually runs a fairly definite history. The ordinary onset is *with pain*. The pain lasts a variable length of time; usually it lasts for months, and there are quite long-drawn-out prodromal symptoms, and pain is a striking feature. Not every case of

tumor is associated with pain, but 99 out of 100 of them will show it. Indeed, if one makes a differential diagnosis upon the basis of the absence or presence of pain in suspected cases of tumor of the spinal cord one will be usually correct. In this case, then, there being no pain, merely a few sensory disturbances, one is compelled to say that the probabilities of tumor are exceedingly slight. That reduces us to the supposition that we are dealing with some inflammatory process.

Now why do we believe it to be an inflammatory or degenerative process? First, you will note the temperature and pulse-rate since he has been here hardly suggest an inflammatory affection. He entered on the 20th of February in a wheel chair. Pulse 70, temperature 97.8° F., respiration 18. His pulse that day was recorded at two other hours as 76 and 80; temperature 98.4° and 96.6° F.

On the following day, February 21st, after an all night's rest, his pulse was 80, 72, 70, and 72, and his temperature varied between 98° and 98.8° F., and his respiratory rate was 20. It is not necessary to go through all of the record for the many days that he has been in the hospital. I will simply state that his pulse usually runs between 60 and 70; his temperature is always normal or somewhat subnormal in the early morning. His bowels have been moved without great difficulty by enemata; no involuntaries whatever have occurred. He does not complain much except of an occasional headache—in fact, he is not the complaining kind. Occasionally he vomits.

On the 22d of February the blood-count showed the leukocytes 8600 and the hemoglobin about 85 per cent. The examination of the urine on the same day showed no albumin; no sugar; no casts; no red cells; two or three white cells in the field, using the $\frac{1}{6}$ -inch objective. On February 20th a similar urinalysis was also made. A lumbar puncture was done by myself on the morning of February 22d and a few cubic centimeters of perfectly normal cerebrospinal fluid were obtained. It contained no lymphocytes above the normal 2 or 3 to a field. A Wassermann examination made upon this fluid was entirely negative. There was absolutely no evidence of inflammation at the time of the

making of the lumbar puncture, so that we may feel quite sure, I think, that a meningitic process is not present.

Now, as to the *physical examination*: First, we will go over the matter of reflexes, which are very important in cases of this type.

First, as to the knee-jerks. You will notice that they are quite increased; a very slight tap, as you see, is capable of producing a very marked jerk. What is true of one knee-jerk is equally true of the other. They are both very much exaggerated and exaggerated equally. When the reflexes are exaggerated to an extent as great as they are here, one immediately wonders whether there is some interference with the inhibition of the reflexes down through the pyramidal tracts, and so, whenever you find knee-jerks of this type, almost the next procedure is to determine whether ankle-clonus is present. In order to do that the knee must be flexed. I simply double up my fist beneath his knee, and then, with the other hand, make a sudden upward thrust of the ball of his foot, and the clonus, as you see, immediately appears. The clonus is a true clonus; it moves back and forth, as you see, in a perfectly involuntary sort of way. So much for the clonus of the right foot, now as to the left foot. It is not excessive, but it is a perfect ankle-clonus.

Ankle-clonus is a symptom about which there has been a great deal of dispute. I believe it is always indicative of organic trouble in the pyramidal tracts. It has been described as occurring in hysteria. True clonus never occurs in hysteria. A very interesting case of this sort was once shown at the Paris Neurological Society, with the diagnosis of hysteria in the presence of ankle-clonus. Babinski on that occasion was of the opinion that the case was an organic one rather than a functional one. The man who reported the case (his name I do not now recall) was honorable enough in the course of a few months later to send a notice to the Review, stating that the ankle-clonus which he had described as functional was organic, because certain indisputable organic symptoms had developed between the time that he had shown the case and the present time. All of the cases that I have ever seen with ankle-clonus were organic; they were never hys-

teric. So now we are justified in saying that there is no hysteria in this boy's case.

Up to this point we feel justified that there is no tumor because of the absence of prodromal pain, and we now find there is no hysteria. We are further sure from the analysis of the cerebrospinal fluid that there is no meningitis.

In these cases where you have ankle-clonus, and where you have exaggerated reflexes on both sides, it is very commonly the case that you find the Babinski reflex present on both sides. The Babinski reflex has exactly the same significance as ankle-clonus; it means trouble in the pyramidal tract; it means trouble anywhere from a cell in the motor cortex down through the pyramidal tract to the anterior horn cells in the spinal cord. The lesion, therefore, may be anywhere from the cortex to the anterior horn, somewhere and anywhere in the course.

If we find the Babinski reflex in this case we have another absolute differential point eliminating the diagnosis of hysteria, because the Babinski reflex does not occur in hysterical states. The reflex is best obtained by stroking, not too severely, with some pointed object—and I usually use the end of a key—the outer plantar surface of the foot; and the result, when positive, is that the big toe instead of turning down, turns up, and it turns up rather promptly. You will notice also the behavior of the other toes. They do either one of two things—they either turn down a little or they spread a little. Babinski has described the latter modification of the toe-phenomenon under the name of “fan sign.” The “fan sign” is simply a spreading of the toes like a fan, and occurs oftentimes very plainly where the extension of the big toe is only very slight. Testing in a similar way the right foot we find also that the big toe turns up in precisely the same manner.

We know now that he has a double Babinski reflex, and we positively know that he has some disturbance in the pyramidal tract somewhere between the brain and the anterior horn. This is the way to work out an examination of nervous cases: Interpret your symptoms as you move along and begin your localization process right away in your examination.

Now there are certain modifications of this Babinski reflex which have been described under other names. One is the Oppenheim reflex. Stroking along the inner side of the ankle here will occasionally lead to a cocking-up of the big toe (demonstrating). In this case it does not. In this case the Oppenheim reflex is not present. Another similar reflex described is the Gordon reflex. Compression of the calf of the leg will sometimes lead to a cocking-up of the big toe. In this case it does not do so. In this instance the Gordon reflex is not present. The Oppenheim and Gordon reflexes are of some value occasionally, because there have been cases recorded where these reflexes have been present and the Babinski toe-sign absent. They have precisely the same meaning as the Babinski reflex, namely, trouble in the pyramidal tracts somewhere between top and bottom.

Now, another important reflex, which is best obtained when the patient can be placed in a kneeling posture, with his feet out behind him, is the ankle-jerk. When the patient is lying down it is not easy to be sure of your findings. If it is found present there is no difficulty. Often, however, it will seem to be absent when it is really present when the test is made with the patient in the kneeling posture. But you will notice here that tapping upon the Achilles tendon while the foot is held in the air by grasping the ball of the foot there is a sharp and exaggerated ankle-jerk. On the right leg the same thing is true.

When a patient is ambulatory the best way to obtain the ankle-jerk is to have him kneel on a chair, his feet out behind him. With a percussion-hammer you then tap the tendo achillis and you can always get the ankle-jerks very nicely if they are present. This test is of great value in cases of suspected locomotor ataxia because the ankle-jerks and knee-jerks in locomotor ataxia are both absent, and it oftentimes happens that the ankle-jerk disappears before the knee-jerk. So that it is very necessary to make this test in suspected tabes.

While we are upon the reflexes, instead of going next to the sensory disturbances, we will continue with the examination of the other important reflexes. First, I will test in regard to the presence or absence of the cremasteric reflex. The cremasteric

is present nicely upon the left side, but upon the right side it is apparently absent; that is to say, I can't get it.

Now, as to the superficial abdominal reflexes, both the abdominal and epigastric: They are easily obtained by stroking (with not too sharp a point) the surface of the abdomen lightly in this way (indicating). You will notice that all four of the reflexes are absent, both reflexes on both sides.

You will notice also that there is a slight amount of dermatographia present. A light line surrounded on each side by a white line is present. That dermatographia is a first-rate indication of a vasomotor disturbance and is ordinarily found in organic cases of the nervous system of a meningitic type, especially in cerebrospinal meningitis. It also commonly appears in functional cases, particularly in traumatic neuroses. You will notice that the redness now is more marked than ever and that little bits of blotches have appeared on either side of the line at a distance one inch away. This is an example of very marked dermatographia. So that, besides the absence of superficial abdominal reflexes and the epigastric reflex, there is also to be noted the presence of dermatographia.

On examining the reflexes of the upper extremity we first test his bicipital jerks. You will notice these also exaggerated. On testing the triceps reflex on both sides you will note that it also is exaggerated. You also note the amount of extensor response of the forearm when we tap over the radius on the radial border of the forearm 3 or 4 inches above the thumb. This reflex is increased on both the left and right side.

Now, as to the jaw-jerks (Patient opening mouth half-way; putting end of pencil lightly on the patient's teeth and then gently tapping the pencil): He has a brisk jaw-jerk. Now, going on with the examination of his reflexes, we ought next to examine the pupillary reflexes. This we have already done, and here in the clinic, where there is a superabundance of light, it is rather difficult. He has a perfect pupillary reaction to light and accommodation; the pupils are equal in size; they show no inequalities of any sort.

So much for the examination of the reflexes. They give to

us one set of facts; namely, that there is something the matter with his upper motor neurons.

We next come to the *sensory findings*: We want to know as to his sensation to touch, temperature, and pain, and something about his muscle-joint sense; whether he has knowledge as to the position of his toes, ankles, feet.

First, *sense of touch*. When sensory disturbances are present they don't have to be found with a magnifying glass. If they are there the patient is aware of it with ordinary and comparatively rough tests. For instance, the touch of the finger is sufficient. (To Patient): Do you feel that? (Touching Patient at foot.)

PATIENT: Yes, sir.

(This was repeated three times and Patient answered.)

DR. MIX: Tell me when I touch you? Each time I touch you please say Yes.

(This was repeated several times and the Patient answered, but not always correctly.)

You will notice that he feels the touch to some extent, but on many occasions at irregular patches over the skin of his leg he does not feel any sense of touch.

Now the next thing we want to know is whether he can distinguish the head of a pin from the point. (To Patient): When I touch you tell me whether I touch you with its point or head.

(This question repeated and Patient answering.)

Now you will notice that his knowledge is about the same in regard to this sense as it was in regard to the touch sense. He got a good many of his answers wrong. I always touched him with the point of the pin, and yet often he answered the head. Not once did I touch him with the head of the pin. This test indicates more of anesthesia to pain in this part of the leg than elsewhere (indicating calf of right leg). (To Patient): Does it hurt? (looping pin through the skin).

PATIENT: Yes, sir. (But the Patient did not appear to suffer as much pain as a person with normal sensation should).

DR. MIX: The looping of the pin through the skin is rather marked evidence of rather severe reduction of his pain sense,

and on removal of the pin there is no bleeding, which is rather characteristic of many of these cases. It is characteristic of hysterical cases as well as of organic ones. (To Patient): On this left foot, does that hurt? (looping pin through skin).

PATIENT: No, sir.

DR. MIX: Can you feel it?

PATIENT: Yes, sir.

DR. MIX: No pain?

PATIENT: Pricks a little bit.

DR. MIX (trying same experiments on other leg): It does not bother him when I put the pin through the skin. This clearly indicates the amount of pain loss. (To Patient): Now close your eyes and tell me which way I am moving your left big toe.

PATIENT: Down.

DR. MIX: Now, which way?

PATIENT: Down.

(This repeated a number of times, the left toe being moved up, down, to the right or left, indiscriminately.)

DR. MIX: Now, you see he does not know a thing about which way his big toe moves. (To Patient): Now tell me which way I am moving this right toe; have I moved it at all?

PATIENT: No, sir.

DR. MIX: I have moved it as far as I possibly can, yet he is not aware of it. The muscle-joint sense is more affected on the right side than on the left.

These muscle- and joint-fibers don't cross on entering the cord; they go up on the same side as that upon which they enter. Consequently, what do we know?

We know that the columns of Goll and Burdach which carry these fibers are more affected on the right side than they are on the left side of the cord. Consequently, we know he has more trouble on the right side of the spinal cord than on the left.

Now as to one other point. You noticed, I think, that the loss of pain sense was a little greater in the left leg than in the right; it was evident to you all that looping the pin through the skin of the right leg caused some pain, but looping it through the

skin of the left leg caused little or none. These fibers that carry the pain impulses decussate upon entrance to the cord, and consequently the greater loss of muscle- and joint-sense on the *right* side and the greater loss of pain sense on the *left* side, both mean the same thing. That is, they both mean greater trouble on the right side of the cord. Let me state it in other words:

These touch and pain fibers cross on entering the cord to the other side; the muscle- and joint-sense fibers go up straight on the side on which they enter; therefore the right side of the spinal cord is more affected, because the right muscle- and joint-sense is more affected, and the left touch and pain sense.

Now the next question is, How high up do these sensory disturbances go? (Touching Patient's abdomen): Do you feel that?

Here again we find the same conditions; that is, there is more of a loss upon the right side than upon the left.

(To Patient): Tell me whether this stroking of the skin of the abdomen upon the left side feels to you just as natural as the stroking upon the cheek upon the left side?

PATIENT: No, sir.

DR. MIX: What is the difference?

PATIENT: Not so distinct.

DR. MIX: Is there any element of tingling in it when I do that as compared with the stroking on the face?

PATIENT: A little bit.

DR. MIX: Is that true also of the right side of the body as compared with the right side of the face? Is there any difference?

PATIENT: Yes, sir.

DR. MIX: What is the difference?

PATIENT: Not so strong. I can hardly feel it on the abdomen.

DR. MIX: We will go a little bit higher and we will see how it is upon the chest. (To Patient): Do you feel that all right?

PATIENT: Yes, sir.

DR. MIX: Is there any difference in the character of the sense?

PATIENT: Yes, sir.

DR. MIX: A little numb?

PATIENT: Yes, sir.

DR. MIX: Tingling?

PATIENT: Yes, sir.

DR. MIX: How is it here, just beneath the clavicle?

PATIENT: Natural.

DR. MIX: Does it feel natural here, about 2 inches above the nipple, as compared with the left side. Is there any difference?

PATIENT: Yes, sir.

DR. MIX: What is the difference?

PATIENT: Not so distinct on the right side.

DR. MIX: Now, taking this point 3 inches above the nipple and comparing it with the sensation on the forehead, is there any difference? It feels perfectly natural here upon the chest?

PATIENT: Yes, sir.

DR. MIX: Very slight disturbances of sensation are not manifested by a complete loss of the sense of touch. When we begin to analyze the sense of touch we find it is made up of at least two or three component parts. One has been named, by Head of London, the "epicritical sense" and another the "protopathic." When we feel touch in the abstract we just are conscious that we have been touched, but we do not know where or by what. This is the primary sensation of touch, protopathic sensation, stripped of all memory associations and judgment. With preservation of the protopathic sense we are merely conscious of the sense of touch, but not where; furthermore, the sensation obtained is a peculiar kind of tingling sensation. When your sense of touch is perfect, in other words, when you have the preservation of both your epicritical and protopathic sense of touch, you have not only the abstract sensation of being touched, but you base judgments and conclusions upon that sense; you know where you were touched, how heavily or lightly, or by what. Hence, if you wish to determine the upper level of involvement of the epicritical sense you must determine when the sense of touch ceases to be natural. There is nobody who has this loss of epicritical sense who will not instantly know what is meant by "Does it feel natural?" I know this personally. I have not got and never will get a restoration of the epicritical sense over the middle cutaneous nerves of my right thigh which were crushed in an automobile

accident. Professor Head worked this whole matter out on his own body. I doubt whether these things would have been discovered had not some one severed his own nerves. This Professor Head did, and so he was enabled to make the discovery that there were such things as protopathic and epicritical senses. Furthermore, I want to call your attention to the fact that when the sense of touch is lost the epicritical sense is the last to be restored.

Now, in these cases if you simply say to the patient, "Do you feel at this level?" and come to the conclusion that the line of demarcation between the levels at which he feels and does not feel is the level of disturbance in the spinal cord, you will be wrong. What you want to do is to ask, "Does it feel natural here?" and go a little higher, "Does it feel natural here?" and find where the line of demarcation comes between the levels where it feels natural and where it does not feel natural.

Now, in this particular instance, you will notice that on his chest the feeling about 4 inches above the nipple is a perfectly natural one. (To Patient): Is it?

PATIENT: Yes.

DR. MIX: But when I come here, about 2 inches above the nipple line?

PATIENT: No, sir.

DR. MIX: It is not natural?

PATIENT: No, sir.

DR. MIX: We have examined him on several occasions, and we have found the upper line of demarcation 2 inches above the nipple line. That line has been fairly constant since he has been here, but not wholly constant. When he entered, the line was about as I have drawn it, about 2 inches above the nipple line on the right side and just about the level of the nipple line on the left, but he has had since that time quite a variation in this level. At one time the level of disturbance was higher than it is at the present time.

Furthermore, there appeared a while back a numbness in his *right* arm and *right* hand. The numbness he complained of involved all the fingers and both sides of the hand. Even at

the present time the hand on both the radial side and on the ulnar has some sensory disturbance. The ulnar side is supplied by the eighth cervical and first dorsal segments of the cord; the radial side by the fifth, sixth, and seventh cervical segments. We know, therefore, that he has had sensory disturbances in his spinal cord extending as high as the level of the fifth or sixth cervical segment on the right side. On the left side there has been no numbness. So we must assume that as far as the right side is concerned there is a disturbed cervical portion of the cord on the right side.

Now we must look at the *motor* phenomena: First, examine the reflexes; second, the sensation; and third, the motor phenomena in examining a nerve case.

First, can he move his legs? (To Patient): Can you move them at all?

PATIENT: No, sir.

DR. MIX: Utter paraplegia. There is not a particle of motion left.

When we come to the examination of the abdomen, we will see if he can move the abdominal muscles.

(To Patient): Draw your abdomen in. Can you pull your belly up like that? He can't do it. He ought to be able to do that perfectly, but he can't do it. In other words, he has lost control of the anterior abdominal wall—of the external and internal oblique and of the transversalis muscles.

The next question is, How much higher does his disturbance extend? Can he use the pectoralis muscles? He can use the pectoralis major on the left side and he uses it also on the right side.

On examining the grasp of his right hand and having him squeeze as tightly as he can it is evident that he has a great deal of loss of power; indeed, he has quite a marked degree of motor loss in *both* hands. Until he was taken ill he was a good big strong boy weighing 142 pounds.

Remember that he came here to be operated upon for a tumor of the cord. After he had been here in the hospital a few days this right hand began to get weak. Suppose he had been operated for a tumor of the cord at the level of the sensory disturbance

which we have marked on the thorax, that is perhaps in the area of the third or fourth dorsal segment. We would not have found anything, and we would have been very much chagrined a few days after the operation to find the right hand beginning to get weak. Yet before our very eyes the strength of the right hand began to fail. The first thing we noticed he quit eating with his right hand and he used his left. Why? Because he dropped things; he dropped his spoon. Now, in the last week or so a change has come. Power is beginning to be restored in this right arm, and he is beginning to lose a little power in his left arm.

(To Patient): Do you still eat with your left hand?

PATIENT: No, sir.

DR. MIX: You eat with your right hand now?

PATIENT: I don't eat with either; I have to be fed.

DR. MIX: The restoration of power in the right hand is very little, not enough for him to be able to eat with it. Whereas, when he first came here it was perfectly easy for him to eat with both hands. He first lost the power in his right hand, so that he fed himself with his left. Now he has lost the power in his left. He is now unable to eat, and he is fed by his nurse.

Now, there is evidently *an ascending process*. It began as an ascending process as far as numbness is concerned. When he entered the hospital on the 20th of February he had power up to his thorax; since the 20th of February he has been having a gradual reduction of power.

Next please notice that there have been no irritative phenomena in the way of neuralgias, cramps, spasms or contractures, and no trophic disturbances. After examining a nervous case, taking up the reflexes, sensory disturbances and the motor disturbances, you ought next turn your attention to irritative and trophic disturbances. The irritative disturbance would have shown itself by convulsions or localized spasms or by twitching, which might take place here and there. Two types of contractures are noted: The sudden clonic affairs or spasms, and the contractures or persistent tonic spasms. None of these are present. There are no contractures of any sort. There are no convulsive movements of any sort.

As to trophic disturbances which are often present in nervous cases, let us first look for muscular atrophies. In his case there is muscular atrophy in the calves of both legs. One of the first things is to determine whether such atrophy is selective, which means the disturbance of the lower motor neurons in some particular location, or whether the atrophy is general. If the atrophy is a general atrophy it is probably due either to lack of use or to paralysis of the upper neurons, so that no messages whatever can get down to the lower motor neurons. That is the type which we find that he has. His calves are equally and generally wasted; his thighs show very little wasting, perhaps some. The loss of the motor impulse to the anterior horn cells has led to an atrophy of disuse of the muscles of his calves. The atrophy is equal on both sides. There is, therefore, as you see, no reason whatever for believing that he has got any disturbance of the lower motor neurons, or any disturbance in his anterior horn cells.

Now there is a disease known as *amyotrophic lateral sclerosis* which is really a combination of progressive muscular atrophy and pyramidal tract disease. That cannot be present in this case because it is a disease without sensory disturbances, which are very prominent in this case. There must be some tolerably acute process present in this boy's case, presumably something which came from the exposure to which he was subjected in January when these disturbances began to appear. Whatever the etiologic factor may be, it must have been rapid in its effect, because between the 19th of January and the 20th of February, when he entered the hospital, he had become completely paraplegic—in just four weeks' time. And between the 22d of February and today, the 16th of March, the loss of power has extended up sufficiently high to involve his arms—not markedly, but to a great degree—so that it is impossible for him to feed himself and he has to be fed by his nurse. Evidently there is some ascending process.

Now, at this point I think it may be well to take out some cerebrospinal fluid and see what it shows. We made one lumbar puncture quite a while ago, February 22d, and we want to make

another today to see whether there is any difference between the one originally made and the findings today. We wish to know whether possibly a change has arisen between the time when we first examined the patient and the present time. It might be also that we will get some information which we do not now possess through this lumbar puncture. (Lumbar puncture made.)

All that is necessary in the way of precaution is to apply iodine to the skin; let it dry for a moment, and then make the puncture. In doing the lumbar puncture all that is necessary is to find the crest of the ilium; locate the spinous processes of the lumbar vertebræ, and going in between the laminæ of the second and third lumbar vertebræ into the spinal cul-de-sac.

Do you notice the spurt? You notice that it is coming out in a little stream?

VOICES: Yes.

DR. MIX: That is altogether too high a pressure.

Now get me another sterilized test-tube, please. (Another tube produced.)

It is under very high pressure. The fluid itself is perfectly clear. We will have it examined. (To Patient): Does your head ache?

PATIENT: Yes.

DR. MIX: It usually makes the head ache for about one-half hour.

Now that pressure (referring to the fluid still flowing) is more nearly as it should be.

In the procedure of a lumbar puncture the main thing is to go in exactly at the right place. Now the next thing is to put on a little bit of collodion to seal the needle opening. Now let the patient lie down; lie on the side for a little while. (To Patient): Is your head aching, Ross?

PATIENT: Yes, sir.

DR. MIX: Ordinarily with the removal of cerebrospinal fluid there is a headache. The fluid is perfectly clear. It does not look as though there is anything particularly the matter as far as superficial appearance is concerned, yet it is under high tension.

There may be, when the laboratory examination is made of this fluid, some information given to us which we very much desire to have.¹

Diagnosis.—It belongs to a type which is rather unusual, but every once in a while we find a case. It is an ascending paralysis with sensory and motor disturbances, and is described under the name of *ascending Landry's paralysis* in most textbooks.

Landry's paralysis, as described years ago, is not a morbid entity. Some cases are meningitis; others, multiple neuritis; some are ascending infections of the cord; some cases are poliomyelitis.

This probably belongs to an infection of some sort which may have been brought about by reason of exposure on the night of the sleigh-ride, or it might have originated even before that, and his numbness which he noticed might have been a consequence of something which took place a day or so before that. There have been no injuries or accidents to his back; nothing to cause any inflammatory disturbance or disturbance from pressure. When you look over his case as a whole you will recall that there is disturbance of the spinal cord in the region of the columns of Goll and Burdach. You know also that in his case both pyramidal tracts are involved; you know further that there is a disturbance of the fibers which carry the impulses of the pain sense. You know further that there is no apparent involvement of the anterior horn cells, at least, that there is no wasting, no atrophy. You have a good deal of knowledge in regard to the case as far as actual deductions are concerned. And further, you know that the process is a slowly ascending one.

The pathologic agents, however, which have brought about these changes we do not know a thing about at the present time. All we know is that he has at present an increase of cerebrospinal fluid that would indicate irritation of the apparatus producing the cerebrospinal fluid, or else some defect in the apparatus which by absorption regulates the total amount of that fluid. In

¹ The subsequent laboratory report was entirely negative, both as to the Wassermann and Noguchi tests, and the presence of an increased number of cells.

other words, at least there is a disturbance in his cerebrospinal pressure equalization apparatus because the pressure is too high.

When the puncture was made the fluid spurted out. It did not behave in that manner when the lumbar puncture was first done. The disease then had lasted only a month. I am disposed to attribute the headache and the vomiting, of which he has lately so much complained, to the increase in this cerebrospinal fluid, and I furthermore believe that after the abstraction of the rather large amount which we have taken today that there will be a marked improvement as far as headache and vomiting are concerned with the release of this intracranial pressure. That the headache and vomiting are not due to an intracranial growth is evidenced by the character of his sensory changes and the absence of choked disk, as reported by Dr. R. J. Tivnen, who examined his fundi.

As to differential diagnosis, you can see it is not a tumor of the cord. The absence of pain at the beginning is against that diagnosis.

It is not a meningitis of the ordinary type; at least there is no evidence that it is a tuberculous meningitis. Why? Because the columns of Goll and Burdach and the pyramidal tracts are involved. Meningitis affects only the meninges. It is no ordinary type of meningitis because of the involvement of the cord tracts, because there is no atrophy, or evidence of the involvement of the anterior horn cells. It is not syringomyelia. It is not a meningomyelitis because the meninges have escaped. There remains for us only one condition, that is

Ascending Landry's paralysis, and that a typical case, coming on acutely; an ascending process, appearing first as numbness of the feet and its manifestations gradually rising in the body until it has reached the cervical cord, followed by motor disturbances. This is the usual history.

Landry's paralysis is really a syndrome. Practically all of the cases fall within one of four groups: the multiple neuritis group, the meningitic group, the anterior poliomyelitis group, and a group of cases left over which cannot be placed in any of the three groups just mentioned.

This is not the multiple neuritic type, because the Babinski reflex is present. We know it is not an ordinary meningitic type because, although the cerebrospinal fluid is increased in quantity, it is as clear as spring water. It is not the anterior poliomyelitis type because of the presence of the marked sensory findings. Very many cases of Landry's paralysis belong to this type, but none of those cases showing sensory disturbances can possibly belong to it. Now there remains a type of Landry's of which I know very little pathologically, in which there is a clinical course very like this clinical course in which you have the appearance of sensory disturbances, then of motor disturbances, gradually ascending and oftentimes, and as we hope in this case, going through a similar set of regressive changes. Just as the process marches up the cord, it marches down again, and we have a restoration of the functions of the various sets of muscles involved, and of the sensations affected. We do not know, however, whether in this particular instance the ascending process has come to a standstill; indeed, it is rather likely that it has not, because whereas a while ago the patient was able to feed himself, he cannot now. If this ascending process goes still higher the time is going to come when it is going to interfere with his respiration. That happens and it leads to respiratory failure. If regression is going to take place it ought to begin before a great while. I have some feeling that he has a little more strength in his right hand than in his left; in other words, that power is coming back in his right hand. That gives us much comfort, because with the increase of strength in his right hand it would seem that regression is beginning to show and that he will gradually do a little better.

So we diagnose the case as *subacute Landry's paralysis*.

NOTE.—May 9, 1915. The patient is still in the hospital. With the release of two test-tubes full of cerebrospinal fluid the headache and vomiting went away never to reappear. Power very slowly returned in his right hand, while his left became so helpless that he had to lift it about with his right hand. In time he began to be able to hold things in his right hand and then to hold a spoon and to eat. The next change was an ability to move his right toes a little and his right leg. Next

he could move it freely and now he lifts it off the bed without trouble.

Meanwhile his left arm has begun to have a return of power. Although he now writes letters freely with his right hand, he can close the fingers of his left hand into the palm only with slight power. Movement is, however, more rapid and natural in the left hand, less faltering and deliberate. And, best of all, he began moving his left leg ten days ago, so that now he moves it very well. He is slowly but surely regaining all of his lost motor power. The sensory gain has even outstripped the motor. No sensory loss is to be found in either arm, and in the legs sensation is almost completely restored except as to the muscle- and joint-sense. There is no doubt of his complete restoration of function. He is getting well as inevitably as he was going down hill in January and February.

Extract from a letter from the patient dated August 17, 1915: Milestone, Saskatchewan. "Dear Doctor: I am feeling fine and able to walk around quite a little without my crutches. But I have got to use my crutches a little yet, but I think in three weeks I will not use them any more for awhile if I keep on gaining the way I am now."

BRAIN TUMOR IN THE RIGHT CEREBELLAR HEMISPHERE

THE patient, forty-eight years of age, Swedish, entered the hospital on the 9th of May. The reason for his being here these sixteen days is that it has been necessary for his slow-thinking family to come to some decision as to what line of treatment they are willing to have followed. On entrance he had 9600 leukocytes, 5,024,000 reds, and 90 per cent. hemoglobin. Urinalysis showed a specific gravity of 1019, no blood, a trace of albumin, and ten or twelve pus-cells to the field, using a $\frac{1}{8}$ -inch objective. An examination made on the 12th showed the same specific gravity, but no albumin and fewer cells. He entered with a subnormal temperature, 96.6° F., and a pulse of 102. His temperature sub-

sequently rose to 98° F., his pulse fell to 74, and since being in the hospital his pulse has remained somewhere around 70 to 80 and his temperature has ranged from 98° to 98.6° F. There has never been any fever, so far as his family can tell us.

He gives an interesting history: He is married to a widow who had two children and by her he has had two other children. She has had no miscarriages by either husband. His family history is negative with regard to tuberculosis and carcinoma. The man has always been well with the exception of pleurisy, of which he has had two attacks. The first occurred six years ago; he had pleurisy with effusion, was sick four months, and had to be tapped twice on the left side. Two years ago he again had pleurisy, but this time it was a "dry" pleurisy and he did not have to be tapped. Remembering that between 70 and 80 per cent. of cases of pleurisy are tuberculous, one has strong reason for believing that his pleurisy was tuberculous, particularly since it was a recurrent pleurisy, and also because, so far as we have been able to learn from his history, there was no apparent cause for the pleurisy. It did not occur as a sequel to some infection, but came on primarily. On account of his mental condition, it is difficult to get from him any accurate description of his symptoms at the time. He is very slow mentally, and I thought at first that this sluggish mentality might be due to his present condition. I find, however, upon inquiring that it has been characteristic of this man all his life to be very slow in understanding and replying to the questions which are put to him. So far as his mentality is concerned his friends inform us that there has been no change.

Present illness: About the 6th or 7th of November, 1914, he began to have headaches which lasted from fifteen minutes to half an hour at a time. A few weeks later he noticed that on getting out of bed he would feel dizzy and would fall back in bed. A vertigo associated with a headache ought to make you very careful in the examination of such an individual. It often means a great deal; it seldom means nothing. It does not mean arteriosclerosis in a man of his age. Very few individuals having hardening of the arteries will complain of headaches; they will com-

plain of dizziness alone. Another thing that is well worth thinking about when you encounter a patient with headache associated with dizziness is that there may be a pontine or cerebellar lesion involving the middle and superior cerebellar peduncles.

At the beginning of his headaches he states that he did not vomit, but he did have trouble with his gait—walking as though he were drunk. In other words, within a period of six weeks from the onset of symptoms we have an association of three very important signs—headache, vertigo, and ataxia—which are three very characteristic symptoms of a certain lesion. The ataxia was of the cerebellar type; he did not sway from one side to the other; he simply was generally unstable upon his feet.

A doctor was called in, and for treatment he was placed upon a liquid diet. If you are ever called in to examine a patient who is sick there is no use to start in with your treatment until you have made a diagnosis. Of what benefit could liquid diet be to him? If you treat before you diagnose you are always going to come out at the little end of the horn. He went to another doctor; this one was wiser and sent him to a hospital and there a diagnosis was made. This was in November. At that time he was having a little trouble with his speech. An *x*-ray picture was taken, but the hospital authorities would not show it to the patient's wife nor to a very intelligent young man, a university student, who is his friend and who is trying to look after him. When the latter asked the people in charge to let him have the picture to show to another physician he was finally told that it could not be found. Confidence was therefore lost in the hospital authorities, and the patient was taken home. Another thing to be learned here: Be perfectly frank with your patient. If you know, tell him, and if you don't know, tell him; never bluff.

After this man went back home the headache and dizziness became worse, and he began to vomit. You would expect the vomiting to be of the type known as projectile, but it was not in this instance. There was always time for his wife to get the bowl to him; he would have a little nausea, he would have time to tell his wife, and she would bring the bowl. By the time he was ready the bowl and towel were ready, and the vomiting usually came

with a good deal of retching on his part. On one or two occasions the vomitus came up very easily. Ordinarily, projectile vomiting is considered suggestive of brain tumor, but in many cases of brain tumor the vomiting is preceded by nausea, and is *not* projectile.

About this time, some two months after the onset, the patient began to have trouble with his eyesight. Sometimes he would see double, but the thing which he noticed particularly was that the pattern in the wall-paper began to fade from his sight. Some of these patients complain of vagueness and grayness in the vision, but he complained of fading of the pattern in the wall-paper.

Now what is the first procedure in such a case? The very first thing is to find out about his eyes, because he has symptoms suggestive of the general symptoms of a cerebral neoplasm: First, he has very severe headache; second, vomiting; third, a disturbance of gait suggestive of a cerebellar lesion; fourth, a dimming of vision and fading of the pattern in the wall-paper, indicating a disturbance of the disks. Consequently, the very day of his entrance we had him examined by Dr. Tivnen, and the report is: "Marked choked disk in both eyes, worse in the left eye; a secondary choroiditis in the left eye." The vision in the left eye was $\frac{20}{200}$; in the right eye, $\frac{20}{30}$, so there is in his case everything we need for the general diagnosis of brain tumor—headache, vomiting, and choked disks.

Localization.—Now comes the much more difficult task of telling where the tumor is. When you make the diagnosis of brain tumor you are just starting. The next thing you have to determine is where it is; next, what it is, and third, how you are going to treat it. In some of these cases the localization is most difficult. Anyone who has done any work in localization knows how difficult it sometimes may be.

Passing up for a moment the subject of localization and considering the subject of treatment, there is this to be said: It is hardly to be hoped that we can remove the tumor if it is found. The chances of removing brain tumors are about 1 in 20. So practically the only thing that can be done in this case, since the

tumor probably cannot be removed, is to save the patient's eyesight and prevent the headaches, and the thing to do is a decompression operation.

The next thing is, Where is the decompression to be done? You will remember that the cranium has two cavities. One lies under the tentorium and contains the cerebellum, pons, and medulla; the other lies above the tentorium and contains the cerebrum. It is perfectly evident that if the patient has a disturbance above the tentorium the thing to do is to relieve the pressure over the hemispheres, and it must be equally clear that if the trouble is below the tentorium any decompression operation above it will not relieve the pressure at all. If the pressure is sub-tentorial the only place to do a decompression operation is back here (indicating the back of the head, below the external occipital protuberance). Hence the very first thing to be decided is whether the operation should be below the level of the tentorium or above it. Suppose we should make a mistake; suppose that we might even say to ourselves that he has a brain tumor, but we can't localize it, so that we will do a decompression over the silent area, the right parietal region; he would be just as badly off after the operation as before. Or, if we thought the tumor was below the tentorium and operated there and found it to be above, the result would be just as bad for him. Consequently, we have just *got* to determine in which of the two chambers the tumor lies.

The localization in his case seems to be comparatively easy; he has had a good deal of vertigo which of late has always been toward the right side. Furthermore, he has had a reeling, drunken gait. This is very characteristic of disturbances of the cerebellum or cerebellar peduncles. There has been another symptom in this man's case which is not in the history and which we would have known nothing about if we had not had him under observation. He has had two or three peculiar "spells." I have not seen him in one of these, and the description furnished by the nurses is poor, but we know that he has spells which are not associated with any convulsive disturbance at all, but with a feeling of intense faintness. There is no complete loss of conscious-

ness, but he inspires a fear on the part of those about him that he is dying. At any rate, the nurse was very badly frightened. The attack seems to have been one of severe vasomotor depression, associated with irritation of the respiratory center. This sort of thing is suggestive of disturbance of the vasomotor and respiratory centers in the medulla, just beneath the fourth ventricle. Furthermore, attacks of this sort associated with respiratory paralysis precede cardiac paralysis. Indeed, in cerebellar tumors and tumors involving the fourth ventricle you can have a paralysis of respiration antedating paralysis of the heart by several hours.

In this hospital we had a notable case of a young girl with a tumor of the fourth ventricle who was kept alive with a pulmotor from Friday night until Sunday morning at 6 o'clock. Her heart was beating all of that time. As soon as the pulmotor stopped she would begin to get cyanotic, but as soon as the pulmotor was started again and the blood was aerated she would pick up; she went on this way for thirty-six hours before she died. In other words, you can have a very great disturbance of the respiration without a corresponding disturbance of the pulse. Careful observations have not been made by the nurses or interns upon this man as to the exact behavior of his pulse and respiration during the attacks, so that I have no accurate knowledge about them. He has the spells very suddenly and they are quickly over. Spells of this sort are very apt to lead to sudden death, and one of the spectacular features of cerebellar tumor is sudden death. They are not convulsive seizures, but belong very distinctly to the type of respiratory center disturbances.

Now as to the examination of the patient: What do we find? Not very much of anything which is characteristic of cerebellar tumors, the only symptoms being the drunken gait, typically cerebellar, the tendency to fall to the right side, and the Romberg sign. As far as his knee-jerks are concerned, they show nothing abnormal; the knee-jerk is reduced a little perhaps, but it is present and alike on both sides. It is so difficult to examine him that you have to use strategy to find out anything. He has no Babinski or allied sign, so that there is no disturbance

of the pyramidal tract. His knee- and ankle-jerks are somewhat reduced, but normal enough; his cremasteric reflexes are present; the abdominal reflexes and epigastric reflexes are not present.

DR. MIX (to Patient): Can you stand up, Mr. B.? Can you manage it? (Patient stands up.)

I want you to notice the way he navigates or, rather, the way he does not navigate.

(To Patient): Now put your heels and toes together and close your eyes.

He has quite a pronounced Romberg sign, as you see. He really can't stand with his eyes closed.

(To Patient): Now, let's see how you walk. (Patient attempts to walk.)

You see he has a tendency to fall to the right, and without support he could not go at all.

VISITOR: Are his muscles spastic?

DR. MIX: No, they are not spastic, neither are they paretic. (To Patient): Give me a grip with the right hand and then with the left.

The grip is about the same with both hands.

(To Patient): Now rotate your forearm back and forth as fast as you can; now do it with both hands. (Patient finds it impossible.)

He apparently has a little more disturbance on the right side. That symptom is Babinski's diadokokinesia; it is a big name, but all there is to it is an inability to alternate rapidly between pronation and supination, and a person with a cerebellar lesion cannot do that on the affected side.

(To Patient): Now let's see the tongue.

The tongue is coated a little, but shows no deviation. There is no paralysis in the face, there is no disturbance in the pupillary reactions, and although there is a history of double vision, yet today he shows no paresis in any of the ocular muscles.

Now in view of the fact that the patient has had pleurisy twice, can it be a tuberculous affair? Can it be a tuberculoma? It is a curious fact that a great many of the lesions occurring before the

age of twenty in the posterior fossa are tuberculomas. This is the statement: Before the age of twenty 80 per cent. of tumors in the cerebellum are tuberculous. We, therefore, gave him some tuberculin to find out if that old pleurisy was tuberculous or if the tumor in his head is tuberculous. We got a negative reaction, which, so far as we know, would rather indicate that it is not a tuberculous proposition. But it by no means settles it, because many of these tuberculomas give a negative tuberculin reaction. Were he a young man the chances of the tumor being a tuberculoma would be extremely great.

There is an old diagnostic rule that but one pathologic process affects an individual. By this rule, if the patient had a tuberculous pleurisy, as we believe, his cerebellar lesion is now apt to be a tuberculoma, particularly since tuberculomas are prone to form in the posterior cranial fossa.

Now you might ask why we did not make a lumbar puncture? What do you think about it? Would it be necessary? The question is not so much the necessity of it as the advisability of it. Why have we not done it? I had a good reason for not doing it. I don't want to take any chances with him. What chances would there have been of trouble? This is the situation: If you have reason to believe that the tumor lies in the sub-tentorial region, when you do a lumbar puncture and let out a lot of cerebrospinal fluid you are liable to permit that tumor to jam the medulla down into the top of the foramen magnum and kill him by its compression. That very thing has happened, and that is why we do not care to do lumbar punctures when we believe there is a large tumor in the cerebellum, medulla, or pons.

Now the last question is, Where is this tumor? We can see that it is not causing paralysis, for he has none. It sounds as though he has some disturbance of speech, but his young friend says that his speech is the same as it has always been. In other words, the testimony of his friends is to the effect that there is no dysarthria; there being no dysarthria, you would be able to say that there is no disturbance of his hypoglossal center in the medulla. We have no evidence of the grosser facial paralyses

which might occur, we have no evidence of any pneumogastric disturbance unless these five-minute attacks, of which he has had three, are to be looked upon as pneumogastric symptoms. It is not a cerebellopontine angle tumor—his seventh and eighth nerves being intact on both sides.

We have left as localizing factors only that he has a Romberg sign, marked static ataxia, that he has well-marked ataxia when he walks—he would fall to the right if he had to walk alone, and he has marked vertigo indicating disturbance of the cerebellar peduncles. There is nothing to tell us on which side of the midline the lesion lies except his tendency to fall to the right. We can be dogmatic about one thing, and that is that the lesion is a neoplasm, probably a tuberculoma, situated beneath the tentorium, and that the thing that has to be done is a subtentorial decompression on the right side. His wife has now come around to the point of yielding, and we shall probably be able to tell you at the next clinic what has been found. If there is anything which can be removed it will be removed. We believe that it is a rapidly growing tumor, for his symptoms began only in November and he is now unable to get about.

There is one last point to be mentioned, and that is that cerebellar tumors are much more apt to give early choked disks than any other kind of tumors. Therefore, we believe that he has a tumor in the subtentorial region and that a decompression operation must be done down to the foramen magnum on *both* sides to give the maximum possible expansion, and if it is possible to recognize and remove the tumor at that time it will, of course, be done, but the chances are very few. The operation will be done largely to relieve his terrible headaches and to prevent the complete loss of sight.

OPERATIVE NOTES ON THE CASE OF BRAIN TUMOR

The case of cerebellar tumor was operated this morning at 9 o'clock by Dr. John B. Murphy. You will remember that this man staggered to the right. Inasmuch as the direction of staggering often records the side of the tumor, it was assumed that the probabilities were that the tumor was in the right cerebellar

hemisphere, affecting the right corpus restiforme. Inasmuch, however, as in these cases diagnosis of the side is frequently wrong, it is always wisest, in doing a decompression, to have the bone removed from the transverse sinus down to the foramen magnum over both hemispheres so that the pressure can be lessened very markedly. In this man's case that was done. On palpating through the dura an area slightly harder than the other area could be felt. There was no pulsation when the cerebellar hemispheres were uncovered. The cerebellum was apparently under great pressure.

On incising the dura at the right the cerebellum began to protrude, but it did it rather slowly, so that it was possible for Dr. Murphy to remove the dura with sufficient rapidity to avoid any possible tearing of the cerebellar laminæ. The left cerebellar dura was also cut, but there was no tendency whatsoever for the left hemisphere to bulge, indicating that the tumor lay wholly in the right hemisphere. The edge of the tumor seemed to fade into the other portion of the brain, so that one felt that one was dealing with an infectious granuloma.

On incising the tumor for the purpose of getting out some of it for microscopic examination it was found to contain some caseous material, thus betraying the infectious nature of the granuloma. We will have a microscopic examination made of the excised tumor.

This operation will relieve his headaches and the pressure upon the floor of the fourth ventricle, and in addition to that it will prevent the further loss of his eyesight. The swelling of the nerve heads during the last week was one-third more. You see how rapidly the trouble was progressing and how even the delay of a day means a great deal.

How shall we treat this man? Precisely in the way that any tuberculous lesion should be treated—cacodylate of soda, syrup of the iodid of iron, and perhaps tuberculin.

My usual plan of treatment is to give 2 grains of sodium cacodylate night and morning. As this preparation is hygroscopic, I prescribe a small quantity at a time, in capsules, with the caution to keep them dry in order to prevent liquefaction.

The syrup of the iodid of iron can be given in 12- to 15-minim doses, in water, after meals; or the method of administration which I favor is to tell the patient to put 6 or 7 drops into each of two capsules, and thus protect the teeth. If the stomach becomes irritable I change to the official ferrous iodid pills, giving

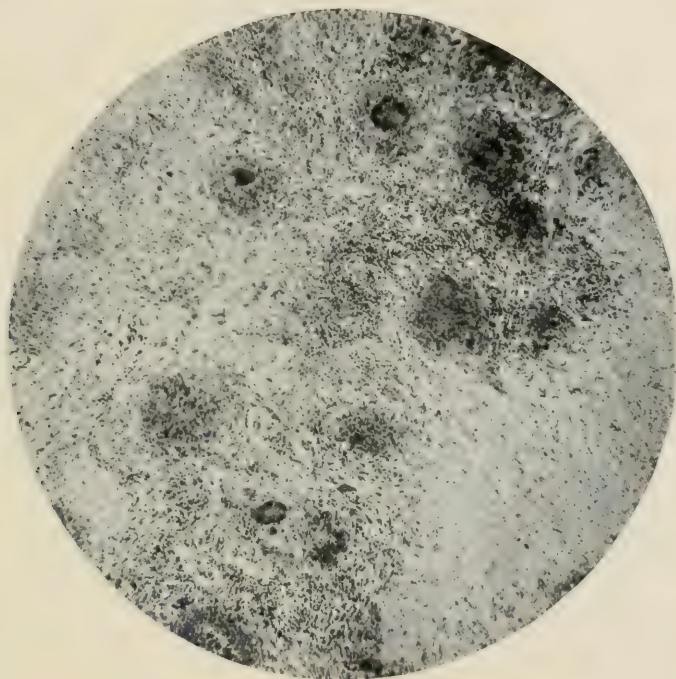


Fig. 96.—Cerebellar tuberculoma. Low power. Shows many miliary and submiliary tubercles with a moderate amount of connective tissue. Giant-cell formation is well marked.

4 daily, for two or three weeks, alternating with the syrup for a like period of time.

The tuberculin is given as in all tuberculous cases. We begin with $\frac{1}{10,000}$ mgm. of Old Tuberculin, giving the injections weekly, the frequency and dosage, of course, being governed by the patient's reaction to the vaccine.

The tuberculin test applied to this patient gave a negative result, but this is extremely apt to happen in cerebellar tuber-

culomas. Though you make the laboratory tests, remember that you are nowhere when you get through if you get negative findings, for tuberculosis and syphilis behave alike, in that they are often present when the tests are negative. We expect this man to improve and we may cure him. Still,

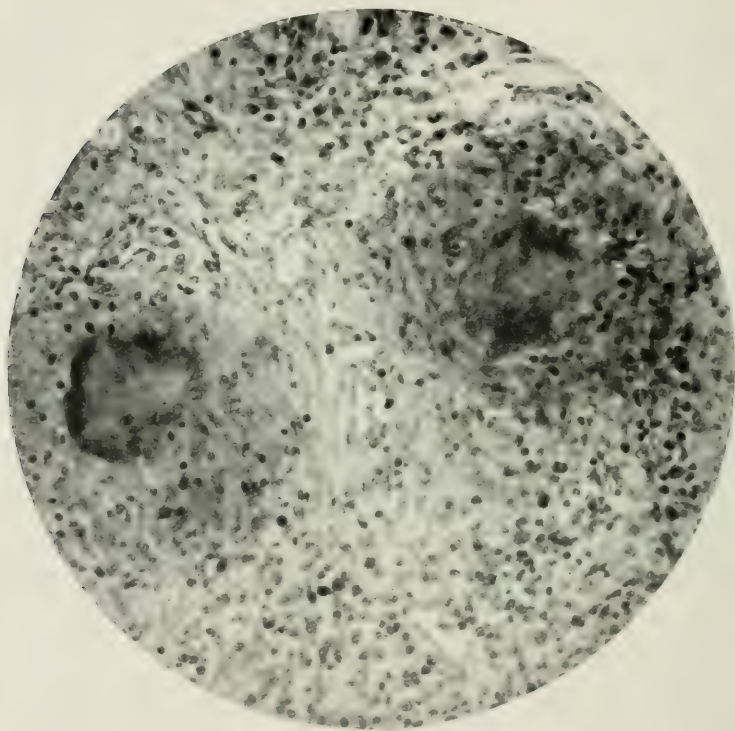


Fig. 97.—Cerebellar tuberculoma. High power. Shows two of the giant-cells with the accompanying round-cell infiltration and new-formed connective tissue.

there is one bad thing about tuberculomas: Where one exists there are apt to be more, and these are apt to grow worse.

NOTE.—The laboratory examination of the tumor showed it to be a typical tuberculoma. The appended plates (Figs. 96 and 97) show sections through the portion excised.

[The patient gradually improved while he remained in the hospital and in four weeks was removed to his home. Though relieved from headaches and vomiting, his eyesight remained unimproved, just as we anticipated. During June the patient's condition remained stationary, but in July he grew rapidly worse, and died about the first of August. No postmortem examination was held, so that we do not know whether the tuberculoma was single or associated with others. Details of his symptoms after he left the hospital are unknown to us.]

CLINIC OF DR. RICHARD J. TIVNEN

MERCY HOSPITAL

A CASE OF BRAIN TUMOR; EYE FINDINGS; DISCUSSION OF CHOKED DISK AND OTHER OCULAR MANIFESTATIONS. THEIR VALUE IN DIAGNOSIS, PROGNOSIS, AND LOCALIZATION.

THROUGH the courtesy of Dr. Mix, I present this patient, a case of brain tumor with ocular manifestations, at our clinic to-day. Dr. Mix referred him to me for an examination of his eye-grounds about three weeks ago.

My findings at that time were:

Vision:

O. D. = $\frac{20}{30}$.

O. S. = $\frac{20}{200}$.

Lids,

Globe,

Tension,

Ocular excursion

Pupils—equal.

} Negative in both eyes.

Reactions:

(a) Direct,

(b) Converg.,

(c) Accom.,

(d) Consen.

} Negative in both eyes.

Fundus:

Marked choked disk in both eyes; most marked in the left; the degree of swelling approximates 4 D. in the left, 3 D. in the right; left fundus also shows some few slight retinal changes in the region of macula.

Fields:

A satisfactory visual field was impossible owing to the patient's inability to concentrate and to his complaint of becoming "dizzy."

I made three subsequent examinations within a period of nearly three weeks. The first and second of these gave practically the same findings as the initial examination. The last examination was made yesterday and showed that the nerve process was exhibiting decided advancement. The left papilledema had increased to approximately 6 D., the right to 4 D.; a few retinal hemorrhages were present in the left fundus, close to the upper temporal margin of the disk; vision in the right had dropped to $\frac{20}{60}$ and in the left the patient had great difficulty in reading $\frac{20}{60}$.

The history of the case discloses that the present illness began last November, a period of six months. The *first* symptom complained of was *headache*. On the history sheet it is noted that within six weeks from the onset of the trouble the patient had developed *headache, vertigo, ataxia, some difficulty in speech, and vomiting*. Approximately seven weeks after the onset of the trouble the first symptom of ocular difficulty appeared—the patient complaining "that the pattern in the wall-paper began to fade from his sight." Summarizing the history, Dr. Mix's findings and my own, the patient presents the following symptoms:

1. Headache.
2. Vertigo.
3. Ataxia.
4. Dysarthria.
5. Vomiting.
6. Marked bilateral choked disk.
7. Marked visual impairment.

The foregoing symptoms are a classic symptom-complex characteristic of a brain tumor, and they, particularly the present state and progress of the choked disk and impairment of vision, indicate clearly that unless an operative procedure is instituted very shortly the patient will become hopelessly blind. This eventuality has been repeatedly explained to the patient and his

relatives, but as yet they are undecided about permitting an operation.

Brain tumor offers a type of disease baffling alike in its problem of recognition, localization, and proper management to the physician, the neurologist, the surgeon, and the ophthalmologist. Only by the concerted, persistent, and painstaking efforts of these investigators—and very often not even then—is this class of cases correctly interpreted and the proper and prompt remedy instituted.

From the ophthalmologic standpoint, the data which the eye examination supplies, in assisting in their recognition and management is of the greatest importance—many times even if this data be only a “negative” contribution. This is so well recognized that no examination of a suspected case of brain tumor can be considered complete or any group of findings therein regarded as conclusive unless in such an investigation an ocular examination be included.

The usual ocular manifestations of brain tumor encountered clinically are:

1. Choked disk.
2. Alterations in the form and color fields.
3. Impairment of vision.
4. Pupillary phenomena.
5. Ocular palsies, diplopia.
6. Nystagmus.
7. Various other phenomena, as alexia, optic aphasia, Benedikt's syndrome, etc.

Of these various manifestations, the most important, the most constant, and the one upon which chief reliance is centered from a diagnostic, prognostic, and therapeutic standpoint is choked disk. Let us discuss briefly these ocular manifestations today, and first as to the choked disk.

Choked disk is regarded by the majority of observers as one of the most constant ocular manifestations and the most common general symptom of brain tumor. Its presence in this condition is variously estimated at from 80 to 90 per cent. Depending upon the views entertained as to the causal factor in its produc-

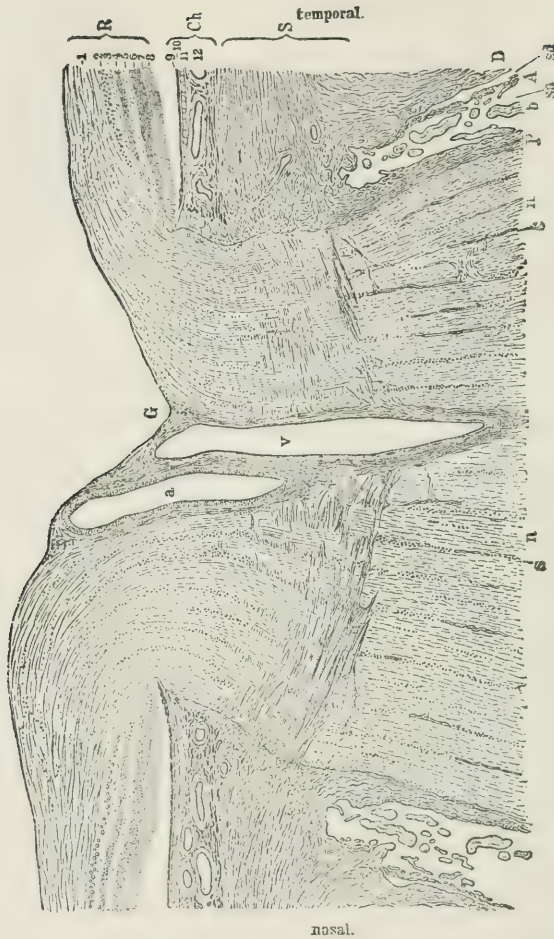


Fig. 98.—Longitudinal section through the head of the optic nerve. Magnified 60×1 . (Fuchs.) In its passage through the scleroticocoroidal canal the optic nerve shows an irregular conical contraction. The fibers of the nerve are collected in bundles, *n*, separated by septa, *s*. Under the form of rows of nuclei, which belong to the neuroglia cells, the continuation of the septa can be followed to the head of the optic nerve. The axis of the nerve is occupied by the central vein, *v*, and the central artery, *a*, which is situated to the nasal side of the vein. The optic nerve is traversed transversely by the lamina cribrosa, which separates the trunk from the head of the nerve. The fibers of the lamina cribrosa arise from the wall of the scleral canal, traverse the nerve in a slightly concave arch (the concavity being directed to the front), and are inserted into the connective tissue that accompanies the central vessels. About at the level of the inner layers of the chorioid the

tion, authors assign the following designation to the phenomenon: "Choked disk," "optic neuritis," "papillitis," "papilledema," and the German "Stauungspapille." Two chief theories are generally accepted as possible explanations of the symptom—the mechanical pressure theory and the toxic or inflammatory theory. Before entering upon a discussion of the relative merits of these theories and also to assist us in appreciating the ocular symptom-complex which these neoplasms frequently present, it will be of value to review briefly the gross anatomy of the retina and optic nerve and their general relation to the brain, its covering, and the surrounding structures. With the assistance of these charts we can readily and rapidly review the anatomy.

nerve-fibers diverge like a sheaf, so as to form a funnel-shaped depression—the vascular funnel, *G*. More fibers pass to the nasal than to the temporal side of the papilla, for which reason the former side is the higher. The fibers of the optic nerve pass over into the fiber layer (1) of the retina. Succeeding this are the other layers of the retina, namely, the layer of ganglion cells (2), the inner granulated or plexiform layer (3), the layer of inner granules or bipolar cells (4), the outer granulated or plexiform layer (5), the layer of the outer granules or of the bodies of the visual cells (6), the limitans externa (7), and the layer of rods and cones (8). The layers of the retina stop short at the head of the optic nerve, the outermost layer, 8, extending the furthest in. The innermost fibers of the sclera, which form the wall of the scleral canal, accompany the optic nerve backward and form its pial sheath, *P*, which is in intimate relation with it. At a point further back from the nerve-head the outer layers of the sclera are reflected backward and form the dural sheath, *D*, which envelops the nerve loosely. Between these two sheaths lies a third, the arachnoid sheath, *A*, which divides the intervaginal space of the optic nerve into the subdural space, *sd*, and the subarachnoid space, *sa*. Anteriorly both end by a cul-de-sac in the substance of the sclera. *b* is the cross-section of one of the numerous subarachnoid trabeculae which connect the arachnoid to the pial sheath. In the wall of the scleral canal is seen the cross-section of some blood-vessels, belonging to Zinn's scleral circle. Between the sclera, *S*, and the retina, *R*, lies the chorioid, *Ch*. The innermost layer of the latter, the lamina vitrea, 10, is the one that extends the furthest in toward the nerve-head, and the fibers of the nerve are constricted by the edge of the lamina. Upon the lamina vitrea lies the pigment epithelium, 9, which belongs to the retina and which on the nasal side extends as far as the lamina vitrea, but on the temporal side stops somewhat short of it. On both sides the pigment epithelium gets to be thicker and more pigmented toward its edge—a state of things which answers to the chorioidal ring that can be seen with the ophthalmoscope. The succeeding layers of the chorioid, the choriocapillaris, 11, and the layer of medium and large sized vessels, 12, do not extend quite up to the optic nerve on the temporal side, because a layer of connective tissue representing a continuation of the sclera juts in between the two.

Anatomy.—The optic nerve might properly be considered an extension or outgrowth of the brain because of its similarity and association of structure and intimate relationship. After collecting the fibers from the retina, it leaves the eyeball, passes through the orbit, and enters the cranium. In studying its course it may, therefore, be divided into three distinct portions:

1. *The intra-ocular termination*, which is in the eyeball or sclera.

2. *The orbital portion*, which extends from the eyeball to the optic foramen.

3. *The intracranial portion*, which extends from the optic foramen to the chiasm.

1. *The Ocular Portion.*—We see in this picture (Fig 98), in its course through the eyeball the nerve does not make a complete aperture through the chorioid and sclera. The innermost lamella of the sclera and a few fibrous bands of the chorioid bridge this area, known as the *foramen sclera*. This bridge is perforated by numerous openings through which the separate funiculi of the nerve pass. This arrangement results in the funiculi of the nerve being separated by numerous septa which form the *lamina cribrosa*. The site of the lamina cribrosa is important clinically, first, because in their passage through this point the nerve-fibers lose their medulla and become transparent; second, being the weakest spot in the eye walls it is the first to give way in increased ocular tension, as in a glaucoma; third, owing to the fact that, at this the only point in its course, the optic nerve is enclosed tightly between firm fibrous walls, it constitutes the vulnerable point where strangulation and constriction may occur as the result of swelling of the nerve. The portion of the nerve in front of the lamina cribrosa is known as the *head* of the nerve or the *papilla*. In pathologic conditions, as choked disk, this portion is raised and projects into the vitreous; normally it occupies the same plane as the retina, or possibly presents a depression, the so-called vascular funnel, which, if of considerable depth, constitutes the *physiologic cup*.

- The Orbital Portion.*—In order to provide freedom of movement for the eyeball the nerve does not describe a perfectly straight course from the eyeball to its point of exit at the optic

foramen, but makes an S-shaped curve or detour. Owing to this provision it is only in such pronounced pathologic states as an exophthalmos from orbital tumor or orbital hemorrhage, etc., that this excess of nerve trunk is put upon tension, and the movement of the globe is, in consequence, restricted. The orbital

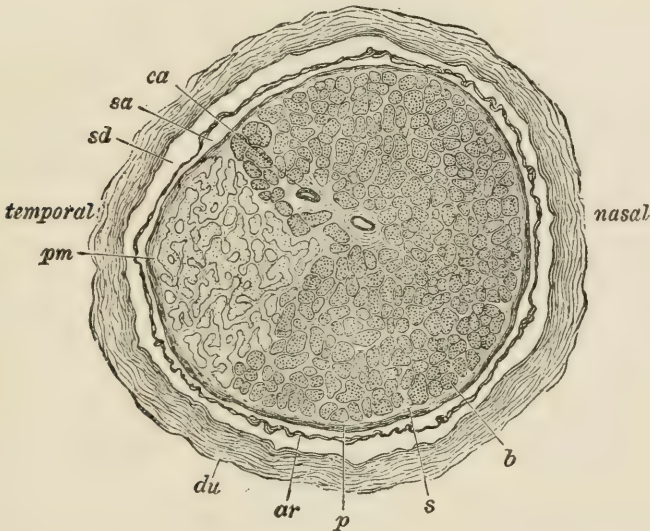


Fig. 99.—Cross-section of the optic nerve, with atrophy of the papillomacular bundle. (Section made 4 mm. behind the eyeball.) Magnified 15×1 . (Fuchs.) The optic nerve is enveloped in the dural sheath, *du*, the arachnoid sheath, *ar*, and the pial sheath, *p*. Between the first and second is found the subdural space, *sd*; between the second and third, the subarachnoid space, *sa*. On the outer and upper side of the center of the section is seen the central artery, *ca*; and more centrally is seen the central vein. These are surrounded by the cross-sections of the nerve-bundles, *b*, which are separated from each other by the septa, *s*, of connective tissue. At the temporal side a wedge-shaped segment, *pm*, is distinguished from the rest of the cross-section of the nerve by its paler color. This represents the atrophic papillomacular bundle. Within the confines of it the cross-sections of the nerve-bundles are narrower, and the septa of connective tissue are correspondingly broader.

nerve trunk is composed of the trunk itself and its enveloping sheaths. It is this particular portion of the nerve and its structure which interests us in our study of the "pressure theory" of choked disk.

The gross structure of the nerve in this locality consists of

nerve-fibers (upward of half a million), separated by neuroglia, and collected into bundles which run parallel with each other and are held together by connective tissue. Lymph cavities exist between the connective tissue and the surface of nerve-bundles and are lined by endothelium. The *enveloping sheaths*, as we see in this cut (Fig. 99) and also in this picture (Fig. 98), are three in number and have their origin in the three cerebral meninges. The *pial* sheath is the innermost and is in intimate contact with the nerve trunk. The *dural* sheath is the outer; it surrounds the nerve loosely and a space is formed between it and the pia—the *intervaginal space*. Within this space lies the *arachnoid* sheath, which divides it into two portions, the subdural and the sub-arachnoid, both of which communicate with the corresponding cerebral channels and constitute lymph-spaces. All these structures and spaces originate in and are continuous with the cerebrum and terminate in the sclerotic coat of the eyeball. The manner of their termination in the sclerotic is as follows: the *intervaginal* space ends by a culdesac in the sclera; the *dura* and *arachnoid* merge into the outer two-thirds of the sclera; the *pia* merges with the inner lamella of the sclerotic and chorioid. Within the nerve trunk are the central vessels (artery and vein) which run in the axis of the nerve to the papilla, where they divide and become the retinal vessels.

The Intracranial Portion.—Referring to this cut (Fig. 100), we observe that this portion extends from the optic foramen to the optic chiasm. The optic foramen constitutes another vulnerable point for the nerve, because it is closely in contact with the bony canal walls, and is easily exposed to such pathologic processes as inflammations, compression, thickening of bone, fracture, etc., involving this locality. After leaving the canal the dura and arachnoid sheaths of the nerve unite with the cerebral dura and arachnoid and the nerve remains only surrounded by the pial sheath. At a distance of approximately 1 cm. after leaving the optic foramen the two optic nerve trunks join, forming the *chiasm*, which rests in the optic groove of the sphenoid bone. From the chiasm they reappear as the two *optic tracts*. The tracts, right and left, proceed backward and diverge to reach the

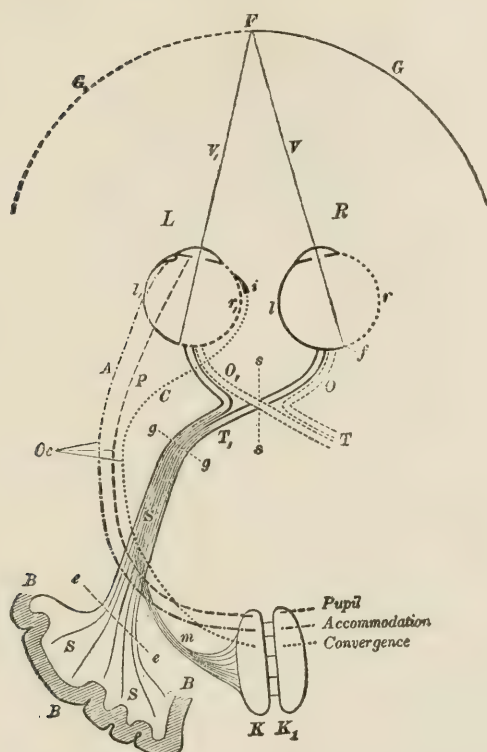


Fig. 100.—Schematic representation of the optic pathway. (Fuchs.) The field of vision common to the two eyes is composed of a right half, *G*, and a left half, *G*₁. The former corresponds to the left halves *l* and *l*₁ of the two retinae, the latter to the right halves, *r* and *r*₁. The boundary between the two halves of the retina is formed by the vertical meridian. This passes through the fovea centralis, *f*, in which the visual line drawn from the point of fixation, *F*, impinges upon the retina. The optic nerve-fibers arising from the right halves, *r* and *r*₁, of the two retinae (indicated by the dotted line) all pass into the right optic tract, *T*, while the fibers belonging to the left halves, *l* and *l*₁, of the two retinae pass into the left optic tract, *T*₁. The fibers of each optic tract for the most part pass to the cortex of the occipital lobe, *B*, forming Gratiolet's optic radiation, *S*; the smaller portion of them, *m*, goes to the oculomotor nucleus, *K*. This consists of a series of partial nuclei, the most anterior of which sends fibers, *P*, to the pupil (sphincter iridis); the next one sends fibers, *A*, to the muscle of accommodation; and the third sends fibers, *C*, to the converging muscle (internal rectus, *i*). All three bundles of fibers run to the eye in the trunk of the oculomotor nerve, *Oc*. Division of the optic tract at *g-g* or at *e-e* produces right hemiopia; and in the former case there would be no reaction to light on illuminating the left half of either retina. Division of the chiasm at *s-s* produces temporal hemiopia. Division of the fibers, *m*, abolishes the reaction of the pupil to light, but leaves the sight and also the associated contraction of the pupil in accommodation and convergence unaffected.

primary subcortical optic centers, the most important of which are the external geniculate body, the anterior corpora quadrigemina, and the optic thalamus. From these primary centers fibers pass to various parts of the brain, but we are concerned in the present instance with but two tracts: First, those fibers which go to the third nerve nuclei which preside over the ocular muscles and pupillary reactions; second, those fibers which pass to the central cortex, the so-called visual area, which have to do with perception.

In the chiasm, as we see at this point (Fig. 100), the fibers from each nerve trunk semidecussate, so that after leaving the chiasm each optic tract contains fibers from both nerves, and hence nerve-fibers from portions of both retinas. This distribution of fibers explains the various hemianopic phenomena frequently present in cerebral neoplasms. It is assumed that "in the lower vertebrate complete decussation of the nerve-fibers takes place at the chiasm; in many of the higher vertebrates a partial decussation exists, the partial character of which is the more pronounced the nearer akin the animal is to man." From a visual standpoint the explanation is offered that in those animals whose vision is monocular a complete decussation occurs, while in those with binocular vision a semidecussation exists.

As a result of the semidecussation of the fibers at the chiasm in man, the *right optic tract* is made up partly of non-decussating fibers from the right half of the retina of the right eye and partly of decussating fibers from the right half of the retina of the left eye; the visual result of this distribution is that the right retinal halves of each eye, which record objects in the left half of both visual fields, are perceived by the right visual areas in the cortex of the right hemisphere. In the same manner the fibers from the left retinal halves of each eye form the *left optic tract*, and the right visual fields are perceived in the left visual area in the cortex of the left hemisphere.

The visual principle is, therefore, enunciated that "everything that the observer sees on the left side of him becomes an object of consciousness through excitation of the right occipital cortex, and vice versa."

Cause of Choked Disk.—As I have said, various theories have been advanced to explain the production of choked disk. Leber suggested an inflammatory theory, under the belief that the intracranial disease leads to the development of phlogogenous material which passed into the cerebrospinal fluid and on to the nerve-sheath, nerves, and papilla; inflammation was set up with edema and infiltration as secondary manifestations. The toxic theory is open to objection, since it has been observed that when a palliative operation is performed and the tumor is not removed the choked disk subsides, an event not to be expected if the toxemia (since these materials would still be present) was solely accountable. Von Graefe's idea was that the tumor caused pressure either directly or indirectly upon the cavernous sinus, thus producing congestion of the ophthalmic vein and central vein of the retina, leading to edema of the papilla and hemorrhages. Schmidt-Rimpler and Manz propose the pressure theory, with the following as its *modus operandi*: In consequence of accumulation of fluid in the intervaginal space a stasis of lymph occurs in the trunk of the optic nerve itself, particularly in the region of the lamina cribrosa, the lymph-spaces of which communicate with the intervaginal space. The edema of the lamina cribrosa causes a compression of the central vessels—compression which makes its influence felt sooner and to a higher degree in the central vein of the optic nerve than in the central artery. As there is constantly pouring into the papilla, through the artery, a quantity of blood which cannot be completely carried away again by the contracted central vein, venous engorgement of the optic nerve and, consequently, swelling of the latter are developed. This swelling of the nerve leads to its incarceration at the spot where it fits so tightly in the foramen sclera, and consequently extreme edema develops in the strangulated papilla.

Fuchs states: "A brain tumor as a result of its growth constantly arrogates more and more space to itself within the cranial cavity; hence, as the skull is unyielding, an increase in the intracranial pressure arises, by virtue of which a portion of the cerebrospinal fluid is squeezed out of the cranial cavity. This fluid finds an egress partly in the direction of the spinal cord, partly

in that of the optic nerve. The spaces between the sheaths of the optic nerve, which communicate with the lymph-spaces between the membranes of the brain, are dilated by an accumulation of fluid (hydrops vaginæ nervi optici).” The manner of this distention and its location and effect are well shown in these pictures (Figs. 101 and 102).

Cushing believes: “It seems in all probability a stasis edema from the forcing of the cerebrospinal fluid into the meningeal

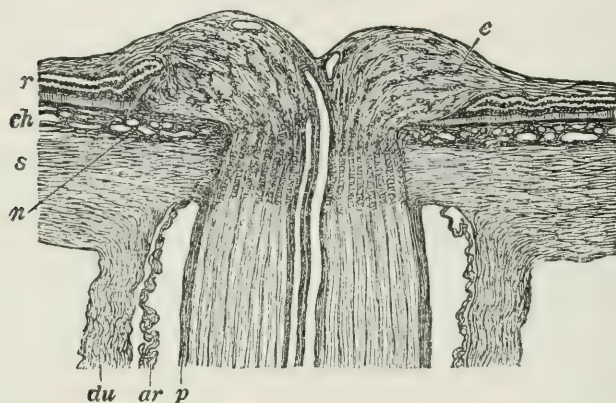


Fig. 101.—Longitudinal section through the head of the optic nerve in papillitis (choked disk). Magnified 14×2 . (Fuchs.) This is greatly swollen, so as to project above the level of the adjacent retina and form at the base an annular tumefaction, the neuritic swelling, *n*. There is a cellular infiltration, particularly along the minuter blood-vessels, *c*, for which reason the latter appear specially prominent. The retina, *r*, is thrown into folds about the circumference of the papilla, in consequence of the swelling of the latter; the choroid, *ch*, and the sclera, *s*, are normal, as is the optic nerve posterior to the lamina cribrosa. Here there is present simply a dilatation of the interstitial space, *i*, through accumulation of fluid, by virtue of which the greatly folded arachnoid sheath, *ar*, becomes especially prominent; *du*, dural sheath; *p*, pial sheath.

sheath which invests the optic nerve. The sheath becomes distended, the nerve-head edematous, venous stasis occurs, and the ophthalmoscopic picture shows, at this early stage, tortuosity of the retinal veins with injection of the disks and more or less haziness of its outlines—the nasal margins usually being the first to become obscured.”

Shieck suggests that "The fluid leaves the meningeal sheath and passes into the optic nerve by way of the perivascular spaces which accompany the central vessels of the nerve." According to Swanzy, the pathologic changes in the papilla "Consist in venous hyperemia, edema, hypertrophy of the nerve-fibers, infiltration of the lymph-cells, and development of connective tissue. Inflammatory changes, although less pronounced, are also present in the trunk of the nerve and sheaths."

It may be concluded that the majority of observers accept the pressure theory as the chief if not the sole causative factor in the production of choked disk, and this conclusion is confirmed

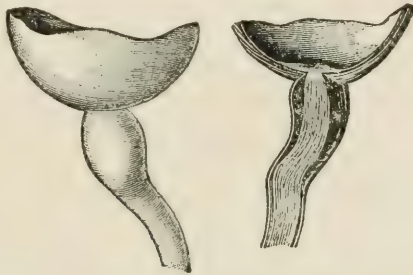


Fig. 102.—Hydrops vaginæ nervi optici. (Fuchs, after Pagenstecher.) Upon the left side is seen the ampulliform swelling of the optic nerve in its anterior portion. Upon the right are represented the relations as seen after making a longitudinal section of the nerve. One can see how the swelling of the nerve is caused by the distention of the outer sheath, which is separated a good way from the trunk of the nerve; and can recognize the projection of the papilla above the level of the retina.

by the observation that the swelling of the disk subsides after a palliative decompression operation.

Ophthalmoscopic Changes.—What are the characteristic ophthalmoscopic changes in the evolution of choked disk? This depends upon the degree of intracranial pressure, how long it has continued, its intensity, the rapidity of the growth, its character, location, and, to a lesser degree, its size. Age may play a minor rôle, as, for instance, in cases of essential or idiopathic hydrocephalus, for in these young subjects, as Cushing says: "Even a low grade of retinal edema is rare, for a pressure sufficient to produce an advanced degree of choked disk cannot well

occur, as the cranium is so easily distensible." Marcus Gunn describes the process in the following five stages:

Stage I.—The earliest ophthalmoscopic signs are increased redness of the disk, loss of definition in its edges, slight prominence of its surface, and narrowing of the physiologic pit.

Stage II.—At a rate which varies much in different cases and which seems to bear a decided relation to the degree of intracranial tension the swelling of the papilla increases, the physiologic pit disappears, and the disk edges become quite obscured; along with these signs there is now slight haziness of the surrounding retina and the retinal veins show evidence of retarded circulation.

Stage III.—In an advancing case the next alteration consists in further swelling of the papilla, so that it becomes more prominent and occupies a larger fundus area, the venous distention becoming very marked; fine folds not infrequently appear in the edematous retina, particularly between the disk and macula, and there may be retinal hemorrhages.

Stage IV.—The papilla becomes more opaque and sometimes more prominent, the hemorrhages increase in size and number, and there are inflammatory exudations on the disk and surrounding retina. At this stage vision has become impaired.

Stage V.—The next stage consists in a gradually decreasing vascularity of the papilla, parts of its surface becoming paler than normal, while the prominence either persists or slowly subsides. At this time also we first note a change in the branches of the central artery in the form of diminished breadth—the state of atrophy with inevitable blindness.

From this detailed exposition of the evolution of choked disk we learn that in this condition, as in all pathologic conditions of the papilla, so far as the nerve head itself is concerned, four cardinal elements must be studied in making the ophthalmoscopic examination. These are:

1. Its color and transparency.
2. Its margins.
3. Condition of level.
4. Condition of the vessels.

With these "cardinal elements" in mind, in summarizing the changes which these stages present, we note as early as Stage I some *swelling of the disk*, loss of definition of its edges, changes in color, and narrowing of the physiologic pit. The subsequent changes exhibit an accentuation of these changes, with the further addition, as the process advances, of marked disproportion in the size of arteries and veins, retinal hemorrhages, and extension of the swelling to the surrounding retina.

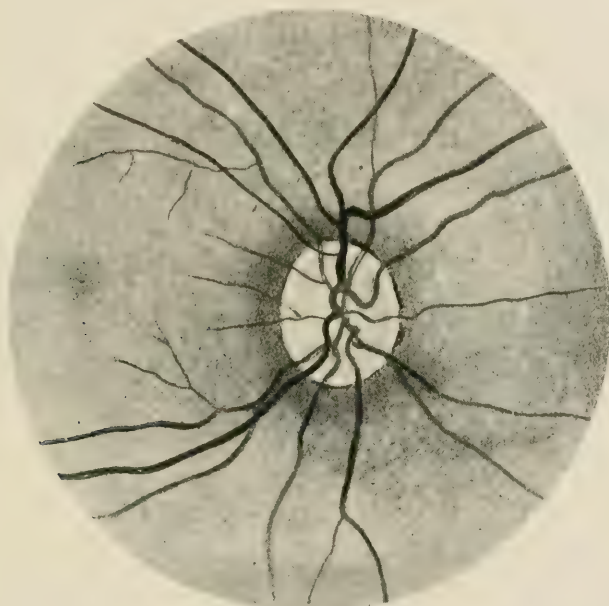


Fig. 103.—Normal fundus of the uniform stippled type. The papilla is oval, has sharply defined margins; is normal in color; the pigment epithelium is concentrated about the papilla and in the region of the macula. The dark, larger vessels, without distinct light streaks are the veins; the bright, narrower ones, with distinct light streaks are the arteries. (From Adam Foster, "Ophthalmoscopic Diagnosis.")

In this plate (Fig. 103) is shown for comparison the normal fundus: The color of the disk, its margins, its level, the size-relation of arteries to veins, all are normal. In this plate (Fig. 104) we see an ophthalmoscopic picture of a well-marked case of choked disk: The color of the disk is markedly changed, its margins are almost entirely obliterated, it is distinctly elevated, the disproportion

between arteries and veins is great, the arteries can scarcely be seen, while the veins are greatly dilated and tortuous. A note on the plate states that the vision in this case was normal, showing



Fig. 104.—Choked disk at its acme. Scarcely a trace can be seen of the margins of the papilla, but the latter seem to send tongue-like processes into the retina. The papilla is distinctly elevated, as can be seen from the course of the vessels, and exhibits a radiating striation. A number of hemorrhages, also striate in form, give the disk quite a specific appearance. The disproportion between the arteries and the veins is so great at the acme that the former are scarcely visible, while the latter leave the papilla as broad, tortuous bands. Some white patches of degeneration are visible in the retina. There are only a few retinal hemorrhages in this case, but they are often much more numerous. The vision in this patient was normal. (From Adam Foster, "Ophthalmoscopic Diagnosis.")

what an advanced stage the neuritic process may assume without impairment of vision. The important and determining change in the disk is the swelling. Von Graefe was the first to direct attention to this change in differentiating an optic neuritis from

choked disk. In an optic neuritis the papilla is usually not swollen to any pronounced degree, and no marked disproportion exists between arteries and veins; the arteries are little changed, while the veins may be dilated and tortuous. In choked disk the papilla is usually markedly swollen and there is great disproportion between arteries and veins; the arteries are smaller than normal and the veins larger.

It is of the greatest importance to estimate the degree of swelling, first, in order to establish the diagnosis; second, to observe its progress. Repeated examinations are often necessary to determine if the edema of the nerve head is progressing, and very often an operative procedure is held in abeyance pending the result of the ophthalmoscopic examination, and it is not unusual for the operative interference to be instituted entirely on the report that such an examination shows marked advancement of the papillitis.

Dr. Mix and myself have at the present time under observation a case of brain tumor, probably tuberculoma, which we have observed for over two years, and which emphasizes the importance of repeated examinations of the disks and the determining character such findings occupy in justifying a certain plan of management. My first examination of this patient's eye-grounds some two years ago gave the picture of an optic neuritis, with only faint swelling; vision in the right eye was normal, in the left considerably impaired; the fields were practically normal in the right, considerable concentric contraction for form and color in the left. He was placed on tuberculous treatment (cacodylate of sodium and syrup of iodid of iron) and all his symptoms disappeared. A year later he had another "outbreak" and practically the same findings were exhibited. He was placed upon the same treatment again and improved to such an extent that he was able to work in his father's store. At this time his condition remains the same; he is under close observation, and should indications justify, an operative procedure will be instituted promptly.

Two methods are chiefly used to estimate the degree of swelling—the refractive and the parallax displacement. In the refractive method a difference in level is calculated in millimeters

from the difference in refraction of the swollen papilla and the retina, a difference of 3 D. corresponding to an elevation of 1 mm. In the parallactic method, using indirect ophthalmoscope, the lens is moved back and forth, and if differences of level exist the fundus details move against one another. An arbitrary rule exists that if the papillary edema exhibits 2 D. of swelling the term "choked disk" is applied.

What effect does the choked disk produce upon vision? The effect upon the visual fields we will discuss later. Referring to Gunn's division we find that vision has not become impaired until Stage IV, when the process is markedly advanced. No definite rule is at hand to serve as a guide, either as to the period of onset of the visual impairment or as to the degree of impairment which may be expected. Choked disk often exhibits a capricious course in its evolution. Only a slight swelling of the papilla may be noted for some considerable period; this may seem to be slowly subsiding, and the other ocular manifestations (vision and visual fields, etc.), together with the general symptoms, showing similar improvement, when suddenly, within a few hours, all the symptoms become accentuated and the papilla presents a marked increase in edema. Our patient today we have had under observation for the past three weeks; for two weeks of this time no noteworthy change was observable in his vision or in the choked disk. This "period of quiescence," however, has ended and at this time we find the neuritic process rapidly advancing and the vision showing the corresponding impairment, all of which indicates that the time for action of a radical nature has arrived.

The plate just shown you (Fig. 104) emphasizes this point, and in my own experience I have many times been surprised to observe the pronounced choked disk present associated with but slight or entire absence of visual impairment. It is a common experience, therefore, to encounter well-marked cases of choked disk with normal vision. This much, however, may be said, that the papillitis appears before the vision becomes affected, and that since the choked disk is not usually a relatively early manifestation of brain tumor, it can, therefore, be stated, with proper reservation, that vision is not, as a rule, impaired early. Patients often

complain of temporary loss of sight occurring several times daily, and varying in length from a few minutes to half an hour.

What practical diagnostic value has choked disk? Its chief value is that it indicates a disease of the brain—usually a tumor. The growth may be in the bones of the skull, membranes, or substance of the brain; it may be a sarcoma, glioma, carcinoma, gumma, tubercle, aneurysm, or a cysticercus, or it may be produced by an abscess, chronic meningitis of the base, hydrocephalus, sinus thrombosis, hemorrhages at the base of the skull, and occasionally it occurs in certain congenital deformities of the skull, nephritis, lead-poisoning, anemia, multiple sclerosis, and certain infectious diseases.

In brain tumor the disk involvement is almost always sooner or later bilateral. Horsley believes that it is apt to appear earlier on the side of the lesion. Marcus Gunn states: "Double optic neuritis with surrounding retinal change coming on quickly suggests the cerebellum; a one-sided neuritis, or marked difference suggests the cerebrum, and, on the whole, is in favor of the tumor being on the same side as the excess of neuritis." It is usually considered, however, that neither the size nor the situation of the growth, the age of the process, or its unilateral intensity are of dependable value in localization; small tumors frequently cause choked disk, while large ones often fail to produce the symptom.

Tumors of the posterior fossa (cerebellum) are the ones with which it is most frequently encountered, and it is more usually an earlier symptom in tumors of this locality than in tumors of the cerebrum. It is said that tumors of the parieto-occipital region, of the corpora quadrigemina and of the cerebellum, show the highest percentage of choked disk; some observers claim that pontine tumors and those of subcortical origin comprise the majority of cases in which the symptom is absent. Cerebellar tumors and those in the midbrain and the thalamus are reputed to produce a more intense choked disk, with rapid invasion of visual function, than those of the cerebrum, the subcortical, parietal, or frontal regions.

The Prognosis from a Visual Standpoint.—As to the prognosis from the visual point of view, it is generally accepted that a well-

marked choked disk, complicating a brain tumor, demands—when other means have failed or are not considered worthy of trial—operative interference, in order that vision may be conserved. The presence of well-marked choked disk not only points clearly to the existence of an increased intracranial pressure, but it also in no uncertain manner calls on the surgeon, other means failing, to preserve the patient's eyesight by relieving this pressure. Locating and removing a cerebral neoplasm which is producing a choked disk and serious visual impairment, while important, must, after all, occupy a secondary place in comparison with what should be the prime consideration in such a case, namely, to restore and preserve the patient's visual function. Horsley lays down the rule that no case of optic neuritis not due to general infectious diseases or to toxic cause should be allowed to go on to blindness without an operation. Through the kindness of Dr. Murphy I have had at various times a number of patients suffering from brain tumor in whom the choked disk was marked, vision impaired, and fields contracted, the process advancing and the visual impairment becoming more pronounced. In these Dr. Murphy has performed a palliative decompression, and the process was arrested and vision either improved or exhibited no further loss. Cushing states that "in the presence or absence of a positive serum reaction or a definite history of lues, antisyphilitic treatment deserves only a brief vigorous trial; if pressure symptoms have been outspoken and do not become distinctly ameliorated in the course of a few days a palliative decompression should be performed and the treatment subsequently resumed should the diagnosis remain in doubt.

Indeed, as a diagnostic test antisyphilitic treatment may be most misleading for two reasons: one because, with certain gliomata, there may be a temporary amelioration of symptoms under these measures; and the other because the fibrous syphiloma, which gives the most characteristic tumor syndrome of all syphilitic processes, is exceedingly resistant, even to massive doses of the usual drugs. Hence, even when the history and a positive serum reaction assure the diagnosis, the lesion must in the end be removed by operative methods if it be localizable, or,

if not, a palliative operation is necessary before the pressure symptoms are sufficiently relieved to allow the patient to receive full benefit from the drug administered. This, too, is the more urgently demanded in case a high grade of choked disk exists. It is certain that in nearly every case, unless relief from pressure is obtained, the optic nerve involvement progresses slowly but surely to optic atrophy and consequent blindness; and though the vision may not show impairment for a considerable period, eventually, if the plus pressure continues, atrophy ensues and soon the color fields become restricted, followed later by marked constriction of the form field, and finally—total blindness results.

Recently I had under observation a case of brain tumor exhibiting advanced bilateral choked disk, visual impairment of considerable degree with marked concentric contraction of the fields. Dr. Mix and myself urged the patient to undergo an immediate palliative operation in order to preserve vision, to be followed later by appropriate medical treatment. This the patient declined; later he became blind and subsequently passed away. This sad eventuality is, as Cushing says, "too often the penalty of procrastination in cases of brain tumor."

Summarizing our facts regarding choked disk and applying these facts in a practical way, we may ask ourselves *What value to the surgeon is the knowledge that the patient has a choked disk?* The answer is that in the present state of our knowledge of choked disk the ophthalmologist is only able to assert to the surgeon that the presence of the symptom indicates, after eliminating other causal factors, that the patient is suffering from a brain tumor. Its presence does not materially aid in localizing the neoplasm. While the rapidity of the onset of the symptom, its progress, its marked accentuation on one side, its unilateral or bilateral ocular involvement, may be more or less suggestive in each individual case, yet it is almost always impossible from these clinical data alone to arrive at any conclusion of value, whether the growth be situated in the cerebrum, the cerebellum, or in other parts of the brain. Neither does the presence of this symptom nor the stage of its development indicate with any dependable and unvarying certainty the duration, the character, or the size of the neoplasm.

Its value in the average case is almost entirely limited to the fact that it points almost invariably, other etiologic factors being excluded, to the presence of a brain tumor. As Bramwell expresses it: "The absence of double optic neuritis does not necessarily exclude the presence of a tumor; but the fact that there is no optic neuritis does suggest doubt; and unless the other symptoms of tumor are very clearly defined or unless the physician feels satisfied that there is no condition present except tumor which could reasonably be expected to account for the phenomena of the case, he will be wise, in the absence of double optic neuritis, to hesitate before committing himself to a positive diagnosis."

Alterations in the Form and Color Fields.—The change which occurs in the visual fields is next in importance to choked disk as an ocular manifestation of brain tumor. Recalling our reference to the anatomy of this region and referring to this picture of the optic pathway (Fig. 100), we note the course of the optic nerve from the retina to the visual area in the occipital lobe, and the course of the third nerve-fiber to the oculomotor nucleus and from the nucleus to the pupillo-ciliary muscle (accommodation) and internal rectus (convergence); we note also the semidecussation of the fibers at the chiasm and their distribution, a portion to each optic tract. An appreciation and correct understanding of these anatomic facts is an absolute essential to the correct interpretation of the effect of lesions located at different points on the "optic highway," and the frequent resultant hemianopic phenomena. Practically all our knowledge regarding the localizing value of hemianopic phenomena is graphically depicted in the picture (Fig. 100). A lesion at *e-e* or at *g-g* (Fig. 100) would interfere with visual impulses from the left halves of each retina, and since these retinal halves preside over the right visual fields of both eyes there would, therefore, result blindness of the right halves of the visual field in each eye—a condition known as right homonymous hemianopsia; in a similar way left homonymous hemianopsia is produced; a lesion at *s-s*, a tumor of the hypophysis, for example (Fig. 100), interferes with the fibers distributed to the inner halves of each retina, which preside over the temporal visual fields of both eyes, and there results blindness of the temporal halves of each visual

field—bitemporal hemianopsia; a lesion, *m* (Fig. 100), obliterates the reaction of the pupil to light, but sight and the associated pupil, accommodation, and convergence reactions are intact;

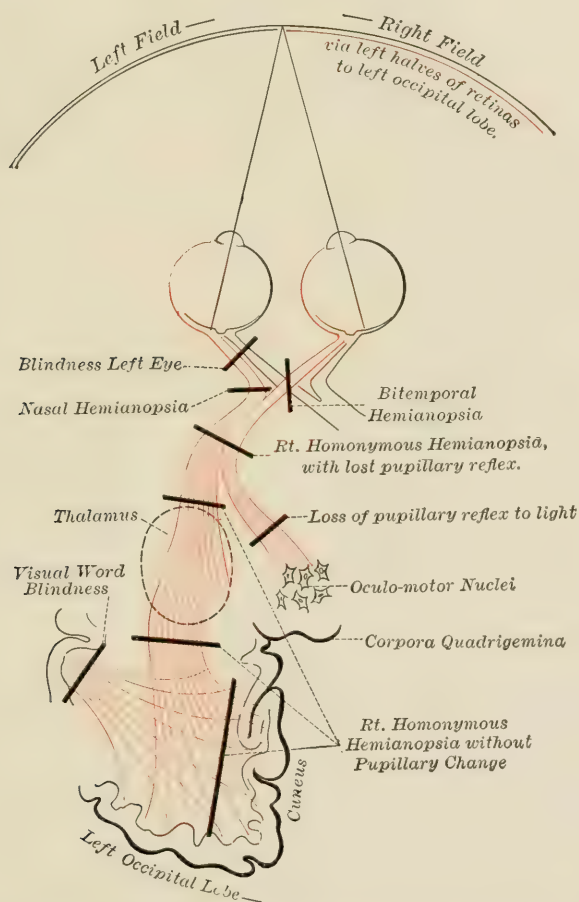


Fig. 105.—Diagram to show various forms of visual disturbance following lesions in different portions of the left visual pathway. (Cushing.)

if only one visual field is involved, or in the case where both fields are affected, but the involvement is not symmetrically situated, the lesion is in front of the chiasm; complete blindness of one eye

with retention of good sight in the other also indicates a lesion is in front of the chiasm; binasal hemianopsia is rare. In this cut (Fig. 105) we see pictured lesions at different areas of the optic pathway and brain and the various hemianopic phenomena resulting, indicated in detail. This picture (Fig. 106) presents a visual field chart characteristic of homonymous hemianopia.

We will now study the various ocular manifestations attendant upon lesions of the different lobes of the brain, utilizing for the purpose Cushing's excellent Summary (Osler and McCrae's Modern Medicine, Vol. VII).

"Frontal Lobe.—There may be cortical epilepsy with movements of head and eyes to the opposite side.

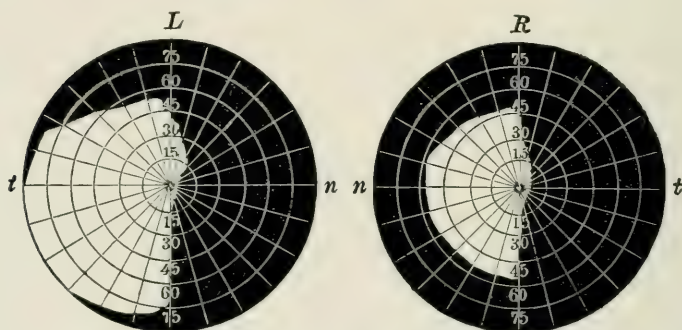


Fig. 106.—Homonymous hemianopia. (Fuchs, after Schweigger.) The areas which have been left white correspond to the left halves of the visual fields, *R* and *L*, of the right eye and left eye, which are still intact; *t*, temporal; *n*, nasal side.

"Temporal Lobe.—Disturbance of the visual fields are common, sometimes as an upper quadrantal homonymous defect; hemianopic visual hallucinations may occur.

"Parietal Lobe.—One characteristic symptom elicited by a tumor involving the left angular gyrus, in the right handed, is the inability to appreciate written language or even to read letters, *i. e.*, word blindness (alexia); a deep-seated lesion of either lobule may involve the fibers of the optic radiation and lead to a half-blindness of the corresponding halves of both retinae, either for colors alone or for form also—homonymous hemichromatopsia or hemianopia.

"Occipital Lobe.—A crossed homonymous hemianopia is the characteristic and may persist for a long time as the only symptom in addition to those incidental to general pressure.

"If optic atrophy secondary to a choked disk has occurred and there is no history of half-blindness before the loss of vision, a regional diagnosis may be impossible. Occasionally the fields for color may be affected before those for form suffer any restriction, and cases have been recorded in which quadrantal blindness has been present, although these fractional defects in the field are possibly more characteristic of implication of the visual pathway farther forward as it traverses the temporal lobe. Homonymous hemianopia, of course, is characteristic of a unilateral lesion of the visual pathway in part of its course, from occipital lobe to chiasm, and neighboring symptoms must be depended upon to determine the seat of the lesion. When the occipital cortex, particularly the more important region of the calcarine fissure, is involved, the loss of half-vision may be ushered in by certain visual hallucinations, such as scintillating scotoma. Involvement of the optic radiation itself on the left side may be accompanied by some of the parietal lobe symptoms just enumerated—alexia or optic aphasia. Lesions farther forward, which include the fibers from the oculomotor nuclei on one side and affect the arc of the pupillary light reflex, lead to the hemianopia loss of this reflex on the blind side (Wernicke)—a condition which is wanting in pure occipital lesions.

"Centrum Semiovale and Basal Ganglia.—Particularly in the left half of the brain the deeper lesions of the centrum lead to very extensive disturbances not only in movement and sensation, but in psychic activity, in the speech mechanism, and in vision. In tumors of the basal ganglia neighborhood symptoms occur, such as oculomotor palsies, crossed pupillary dilatation, ptosis, and hemianopia.

"Brain Stem (Crura, Corpora Quadrigemina, Pineal Gland, Tegmentum, and Pons).—Several examples of tumors of the crura, often a solitary tubercle, have been recorded, the characteristic symptoms being a contralateral hemiplegia with homolateral or bilateral oculomotor palsies. Involvement of the corpora

quadrigenina leads to failure of sight, to nystagmus, to persisting palsies of the ocular movements, particularly those in a vertical direction, with preservation of lateral movements, and probably to loss of pupillary reflex to light and even to convergence, as the centers for the oculomotor nerves become involved. The pupils may be irregular and Bielschowsky describes a form of nystagmus with clonic twitching. Thus a combination of ocular paralyses and ataxia (Nothnagel) especially characterizes lesions in this situation, whether they arise in the pineal gland or corpora quadrigenina. When the tegmentum is implicated, oculomotor palsies (similar to those occurring in pineal gland and corpora quadrigenina) commonly occur, and a characteristic paralysis of conjoint ocular movement toward the side of the lesion. In the pons certain forms may show abducens palsy. Abducens palsies must be scrutinized carefully, however, as they are the commonest of all cerebral nerve lesions, and may, from pressure or from stretching through dislocation, accompany a tumor in almost any situation. Neighborhood symptoms are shown, if the growth presses forward, by oculomotor symptoms. In pontine lesions a choked disk is notably late in its appearance or possibly may seem to be so, owing to the fact that the diagnosis of a lesion in this situation is apt to be made early.

"In tumors of the fourth ventricle choked disk is rare.

"Cerebellum.—Choked disk is supposed to be found in a much higher percentage than is the case with growths elsewhere, probably due to the fact that in the course of time a cerebellar tumor will almost certainly lead to an obstructive hydrocephalus, in consequence of which neuroretinal edema is inevitable and may occur abruptly, often with a rapid augmentation of general pressure symptoms. In the absence of the complication and with a slowly advancing benign lesion, choked disk may be absent for years. In extracerebellar tumors a peculiar triad of symptoms has been pointed out by Oppenheim, namely, disturbance of hearing, paralysis of winking, and diminished corneal reflex. The abducens may suffer, there may be weakness of the conjugate movements of the eyes, and nystagmus. The ocular movements are also affected in ways characteristic of cerebellar diseases.

"Cranial Base.—The cavum Meckelii is not an uncommon seat for tumors which may give all the pain of a severe trigeminal neuralgia during the process of stretching or destruction of the Gasserian ganglion, and keratitis is a frequent accompaniment of the process.

"Hypophysis Cerebri.—Any mesially placed tumor in the interpeduncular space naturally presses upon the optic tracts or chiasm, leading in the majority of cases to partial amblyopia and to primary optic atrophy. This in most striking forms occurs as a bitemporal hemianopia, although by no means is this the only form of perimetric defect, for a homonymous hemianopia may occur or one nerve may suffer much more than the other, so that, for example, total blindness in one eye may be associated with normal vision or some stage of hemianopia in the other. Choked disk is usually absent, as there commonly is no great increase in intracranial tension, although it may occur later should the growth enlarge so as to push its way into the third ventricle and obstruct the foramina of Monro."

Dyschromatopsia.—This condition, variously known as an imperfect discrimination of colors, incomplete color-blindness, interlacing and inversion of the color fields, was formerly thought, principally through the writings of Cushing, Bordley, and Heuer, to be an early and frequent indication of plus intracranial pressure preceding even the recognizable disk changes. Later and more perfect perimetric observations, however, have determined these investigators to abandon this view. The visual field in choked disk exhibits many vagaries; in the beginning it may be and usually is normal; later, concentric contraction for form and colors, scotomas, and various hemianopic phenomena occur. The *pupillary findings* in the brain tumor are not, as a rule, from a diagnostic and localizing sense, of great value. The Wernicke hemiopic pupillary reaction may be of service in these cases presenting homonymous defects, usually hemianopia in the field of vision; in these cases, if, when the blinded portion of the retina is illuminated (direct light reaction), the pupil fails to react, it would indicate that the lesion is located in the optic tract below the point where the oculomotor fibers are given off; if pupillary reaction is

present it would indicate that the lesion is above this point. This pupillary phenomenon is of great value when it is present, but the technic is somewhat difficult.

Differential Diagnosis.—We have already discussed the differential diagnosis of an optic neuritis and a pressure neuritis or choked disk. Exceeding difficulty is many times experienced in differentiating cases of nephritis from brain tumor, not only in their general manifestations, but more particularly in the changes the eye-grounds present. Bramwell, Bordley, and Cushing believe that “albuminuric neuroretinitis” is produced in the same way as tumor choked disk, namely, by pressure. Fuchs states that a special form of neuroretinitis occurs in brain tumor which presents, in addition to the usual papillary changes, minute splashes of silver luster in the region of the macula, so that a picture resembling that of albuminuric retinitis is produced. Our case to-day exhibits a retinal change in the left macular region of this character. The general symptoms, particularly the urinary findings, are in such cases exceedingly valuable in differentiating.

Headache.—This perhaps is not properly an ocular manifestation, but it is so popularly regarded as due to the eyes that many of these patients consult the oculist first for relief, and the examination discloses the fundus condition. It is presumed that in tumor, nephritis, etc., the pain is due to the plus dural tension. The headaches vary in intensity in individual cases and they may be referred to any locality of the cranium. Their localizing value is not dependable and only occasionally suggestive.

Other Manifestations.—Cushing has directed attention to engorgement of the veins of the scalp and the smaller venules of the eyelids as a symptom occasionally present. It may be mentioned that the external appearance of the eyeball gives no indication whatever of the deep pathologic process, but presents a normal appearance. We have now discussed the more common ocular manifestations of brain tumor, the frequency of their exhibition, and their localizing and other value to the neurologist and the surgeon.

Summarizing the main facts in this consideration in a practical way we are justified in concluding:

1. That one of the commonest and most dependable symptoms of brain tumor is bilateral choked disk.

2. That when bilateral, the value of this symptom to the surgeon is mainly confined to the conclusion that its presence indicates in the great majority of cases a brain tumor; but that the presence of the symptom in one or both eyes, its unilateral or bilateral intensity, etc., is in most cases of no dependable or unvarying value either in localizing the growth, indicating its character, or estimating its size; its unilateral or bilateral presence, however, together with the character of its evolution, etc., has in certain instances a limited localizing value, and frequently the character of the evolution and its intensity is a determining factor in instituting an operative procedure; so common is this experience and so capricious is the evolution of the papillitis in many cases that all obscure cases of suspected brain tumor and all cases exhibiting the slightest involvement of the papilla should be subjected to repeated ocular examinations.

3. That since vision in a great number of cases of brain tumor is normal and in many instances continues so, even though the pathologic nerve process is far advanced, this fact should not, in the presence of other suggestive symptoms, lead to the conclusion that the eyes are not affected; this is the more emphasized when it is recalled that choked disk is relatively a late symptom of brain tumor and the external appearance of the eyes themselves are normal and give absolutely no hint of the grave process developing in their deeper tissues; the safe rule in all suspected or obscure cases is an examination of the eye-grounds.

4. This class of cases emphasizes the value of co-operation on the part of the physician, the neurologist, the surgeon, and the ophthalmologist; it is often impossible to clear up an obscure case of this character without such co-operation. In order that these investigators may obtain the maximum degree of co-operation and also that the various numerous and complicated investigative methods of technic required produce the greatest efficiency, correctness, and accuracy, these patients should be made hospital cases.

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